ISSUED BI-MONTHLY. VOL. XLVII., No. 2

ARCHIVES

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MARCH

NEW YORK
G. P. PUTNAM'S SONS, 2, 4, & 6 WEST 45TH STREET
AND NEW ROCHELLE, N. Y.
LONDON: 24 BEDFORD STREET, STRAND
WIESBADEN: J. F. BERGMANN'S Verlag
PARIS: J. B. BAILLIÈRE, 19 Rue Hautefeuille
1918

Entered at the Post-Office, New Rochelle, N. Y., as Second-Class Mail Matter

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ARCHIVES OF OPHTHALMOLOGY.

DISTORTIONS OF THE VISUAL FIELDS IN CASES OF BRAIN TUMOR

(*Fifth Paper*)

CHIASMAL LESIONS, WITH ESPECIAL REFERENCE TO HOMONYMOUS HEMIANOPSIA WITH HYPOPHYSEAL TUMOR.[1]

BY CLIFFORD B. WALKER, M.D., AND HARVEY CUSHING, M.D., BOSTON.

(*With seventeen figures in the text and two illustrations on Text-Plate VIII.*)

IN the preceding paper of this series especial stress was laid on the bitemporal defect of vision associated with tumors in the interpeduncular space, particularly on those of hypophyseal origin. It was then stated that in a series of 148 patients, observed up to July 1, 1914, with outspoken manifestations of hypophyseal involvement—hyper- or hypopituitarism—101 had shown neighborhood symptoms of tumor, and of these 101, the visual pathway in 81 had become implicated.

These 81 cases, furthermore, were subdivided into three groups in accordance with the distortions of the fields at the time of the patients' admission to the hospital: (1) those exhibiting a typical bitemporal hemianopsia or a tendency in that direction (26 cases); (2) those exhibiting a typical homonymous hemianopsia or a tendency in that direction

[1] From the Peter Bent Brigham Hospital, Boston, Mass.

(12 cases); (3) those in which complete blindness in one eye was present on admission, the other eye either being involved not at all or showing some constriction of nasal or temporal field (35 cases); and (4) those which presented bizarre fields of indefinite and irregular outline which were impossible to satisfactorily classify (8 cases).

In the succeeding three years to July 1, 1917, there have been 123 additional cases. The entire series at present represents 271 cases, of which 183 showed tumor manifestations and 148 showed field disturbance. This disturbance was bitemporal in 47 cases and homonymous in 22 cases. The remaining 79 of those showing field disturbances were blind in one eye, so that it could not be positively determined to which group they belonged.

From these statistics it is notable that bitemporal defects occur only about twice as often as homonymous defects in this group. That is, of the 148 cases showing field defects 14.8 per cent. are definitely homonymous in tendency, while 31.7 per cent. are definitely bitemporal.

It is with the twenty-two cases exhibiting definite homonymous defects, together with three cases in the amaurotic group, which were known from the history to have originated from a lateral rather than from a mesial implication of the chiasm, that this paper will deal.

Although attention has been called to the fact by others that with pituitary tumors of one sort or another homonymous hemianopsias may occur, it is nevertheless regarded as an unusual consequence of these lesions, and few ophthalmologists or neurologists venture upon a diagnosis of a lesion in this vicinity in the absence of a bitemporal defect, and indeed unless the bitemporal defect is fairly symmetrical.

In our preceding papers[1] especial emphasis was laid upon the character of the advance of the field defects toward the textbook type of bitemporal lesion, with vertical meridian bisecting the field, and it was shown that the advance is a fairly characteristic one, beginning, as a rule, in its earliest form

[1] Cushing, H., and Walker, C. B.: "Distortions of the Visual Fields in Cases of Brain Tumor," *Brain*, 1915, xxxvii., 341.

Walker, C. B.: "Contribution to the Study of Bitemporal Hemianopsia," ARCH. OPHTH., 1915, xliv., 369.

with an upper temporal loss of colors or very small visual angle test objects and progressing until merely an excentric patch of vision remains in the nasal field before blindness occurs.

Bitemporal hemianopsia was regarded therefore as a condition characterized by progressive changes in the temporal peripheries rather than by any particular fixed type of field defect. This seemed the more necessary for the reason not only that homonymous defects were almost equally characteristic of hypophyseal lesions, but for the reason that in many instances it was almost impossible to tell, given a certain stage and without the opportunity to observe fluctuating conditions by a series of charts, whether the condition actually was approaching or had gone through a bitemporal or an homonymous change.

Thus with an upper temporal defect in one eye and normal vision in the other, either type of bilateral field defect may follow: on the other hand, with an upper temporal defect in one eye and with the disturbance in the other gone on to blindness, either type of bilateral field defect may have preceded. Some of the better examples of these things occur in this series of homonymous distortions, and we were at first tempted to include them in the same report with the bitemporal cases.

On the other hand, a nasal defect in one eye gives much better presumptive evidence of an incipient homonymous defect though it may be difficult to determine whether it is of anterior or posterior origin with respect to the primary centers.

Here we would be greatly assisted in making a differential diagnosis if the Wilbrand prism test and the Wernicke pupillary reaction test could be used according to their theoretical conception. But as we have shown in previous communications,[1] the Wilbrand test is not so much dependent on the existence of a definite reflex arc as upon habit formations and psychological phenomena with respect to ocular movements

[1] Walker, "Topical Diagnostic Value of the Hemiopic Pupillary Reaction and the Wilbrand Hemianopic Prism Phenomenon," etc., *Journ. of the A. M. A.*, 1913, Pt. 2, vol. lxi., p. 1152; "Observations on the Topical Diagnostic and Psychiatrical Value of the Wilbrand Test with a New Clinical Instrument," ARCH. OF OPHTH., 1915, vol. xliv., No. 2, p. 109.

with hemianopsia present. Accordingly we consider the Wilbrand test unreliable. The Wernicke test or hemiopic pupillary reaction also has failed to come up to expectations. We have found the hemiopic pupillary reaction present in all cases of anterior hemianopsia where the intrinsic pupillary mechanism was functioning well, but also it has been found present in hemianopsias of known posterior origin. The reaction may often be somewhat less marked in posterior than in anterior lesions, but in only one or two cases in a large series has the hemiopic pupillary reaction been absent with good intrinsic pupillomotor mechanism. Possibly it is true that most posterior lesions disturb the primary centers sufficiently to provoke the reaction so that it is only absent in isolated cases of the most uncomplicated type of posterior lesions.

Further light on the differential topical diagnosis may be supplied by certain characteristics of the field defects to an extent proportional to our knowledge of the mechanism of production of such defects. A few remarks on this subject before the presentation of cases may aid in the discussion.

MECHANISM OF CHIASMAL HEMIANOPSIA.

A very useful device to study the effects of various disturbances about the chiasm may be made by tearing away the strands of a piece of gauze until only a dozen or so fibers are left interwoven at the center. These may be taken to represent the crossed fibers and colored differently from other fibers laid along each side to represent the uncrossed fibers. The study of strains and tensions at various points in such devices, together with the study of autopsy specimens and X-ray, has led us to certain ideas about the action and reaction of forces in the chiasmal regions.

To take the simplest case first, we may consider bitemporal hemianopsia due to a symmetrical struma of the hypophysis. Here pressure is exerted on the lower part of the chiasm usually rather behind the mid-portion, so that the chiasm may be elevated and ride saddled into the upper anterior surface of the growth. An upper temporal defect begins, but does not cross the vertical meridian, although the nerve fibers representing each side of this meridian must be exposed to the same pressure

from the growth being practically equidistant from the same. Yet the crossed fiber suffers while the uncrossed fiber escapes. Hence pressure alone does not account for the result. Tension and strangulation of crossed fibers, because of their interwoven condition in their decussating region, are the additional factors. This is well seen in the model when a similar pressure is made. The crossed fibers are put under tension and strangulate each other, while the uncrossed fibers are free to move away slightly, thereby receiving practically no tension or pressure, although lying possibly side by side with the crossed fibers. The tension of course augments the pressure and vice versa, and both increase the strangulation effect on the interwoven crossed fibers. Indeed it would seem that the strangulation action really affects the physiological block because we have seen the tracts themselves considerably stretched without being blocked correspondingly in function.

This process continues until complete bitemporal hemianopsia results, and then the progress of field failure may hesitate for some time. This hesitation simply represents the amount of additional pressure or distortion necessary before the uncrossed tracts may be in some way affected by the increasing tumor. It is evident from our specimens that several mechanisms and several types of field failure may then take place. The relations of the anterior clinoid processes and the tough dura stretched between them may act as points and lines of counter pressure against which the nerves are pressed or dragged. The remaining nasal field more often seems to shrink a little faster from below than from above, as might be expected to result from pressure upward against bone and dura of the foramen. There is no definite rule, however, and the nasal field may shrink up in a variable manner or quite symmetrically.

While these general considerations may account for much of the phenomena of bitemporal hemianopsia there remains to be explained the early occurrence of scotomata of the cæcocentral type and the temporal island which we have previously described.[1] The former may be the result of hypersensitiveness or possibly toxic œdematous action or both. The only

[1] Walker, C.B.: "Contribution to the Study of Bitemporal Hemianopsia," ARCH. OPHTH., 1915, xliv., p. 369.

explanation of the temporal island we have to offer is that it may result from the "knee"[1] of crossed fibers which loop far forward to the opposite side of the chiasm. Such a loop would act to relieve the tension and strangulation which occur in other crossed fibers of more direct course, thereby preserving vision slightly longer in the part of the field represented by the loop or knee fibers.

The growths producing the bitemporal defects are usually of strumous character and not so hard, so large, or so rapidly growing as the tumors producing homonymous hemianopsia. If, however, the tumor is of firmer and more irregular nature it may lean decidedly toward one tract or the other. Of course if this occurs after bitemporal hemianopsia has been produced, we have finally blindness in one eye and hemianopsia in the other, so that it may be difficult to tell in what class of hemianopsia the condition belongs excepting if we have records or history of the onset. Such tumors may oscillate to a considerable extent so that one part of the field improves as other parts are damaged. Indeed we have one case that started with an early bitemporal defect which finally became homonymous, the temporal defect in one eye recovering as the tumor broke through its confines laterally and destroyed the tract.

From such points as these it is evident that we may estimate whether the tract is being damaged by a growth substantially to the outer side of the tract or to the inner side. Thus if the tracts are affected in the former manner the homonymous hemianopsia is apt to begin and perhaps always be more advanced in the nasal field, while in the latter case the onset is in the temporal field, possibly both temporal fields, and perhaps only later and more slowly is the homonymous nasal field affected.

It is evident that the conditions so far discussed may be fairly well defined, but in addition we find numerous variations even to the most bizarre, resulting from tumor invasion of this region. Sometimes when the variation is not too marked we can estimate the probable mechanism, but then again we may find at autopsy a distortion of chiasm, tract, and nerves so

[1] Wilbrand and Saenger, "Die Neurologie des Auges," vol. iv., p. 147.

marked that it is perhaps only surmise to speak of definite mechanisms. Several factors may be found superimposed. In general it may be said that a great variety of distortions are possible about the region of the optic foramina and clinoid processes. The nerves and chiasm may have the appearance of being strangulated, pulled, or pressed at numerous points, any one or various combinations of which might produce the resulting field defect. At the same time, in extensive tumors the tracts and primary centers may be greatly distorted, giving additional possibilities for field defects.

From a study of our specimens it would seem that pressure immediately lateral to the chiasm is not nearly so effective in producing homonymous hemianopsia as pressure on the tracts farther back against the peduncles. Indeed the sharply homonymous field defect seems always to indicate tract lesions in this group of tumors, while the more anteriorly the tumor presses, the more bizarre may be the departure from the typical picture as the chiasm and nerves are distorted.

However, the chiasm and anterior portion of the tract may suffer marked distortion without showing marked field defect. Such an instance may be now presented in accordance with our plan of presenting the cases in the order of magnitude of the field defect. All the cases in the homonymous group cannot be presented in this paper, but only such instances as seem to show the more typical characteristics of the group.

CASE 1 (Surgical No. 61). *Acromegaly with tumor. April 16, 1913.* Mr. G. B. S., aged 34. Onset of acromegalic overgrowth between 20 and 25 years of age. Sugar in urine first noted six months ago. Polyuria noted for two years. Headache in morning for years, increasing recently. Impotence began three years ago, complete one year ago. Drowsiness two years. Occasional nausea and vomiting last six months. Disposition very irritable recently. Petit mal attacks during last year. There has been no subjective disturbance of vision whatever.

Examination on Admission: Typical example of well-advanced acromegaly. Eyes perfectly normal in appearance and function throughout. Fundi normal; lamina cribrosa clear in good-sized optic cup. No venous engorgement or tortuosity. No disk œdema. Fields of vision perfectly normal to 5*mm* disks, form and colors. Sella turcica shows a tremendous enlargement by X-ray.

April 29, 1913. Operation: Transphenoidal sellar decompression with partial extirpation of struma accomplished without difficulty. The microscopic examination showed a different structure from other tumors of the series. The cells were more spindle-shaped and more compacted.

Patient improved subjectively after the operation and returned to work in very satisfactory condition, but finally was brought back to the hospital, *Mar. 13, 1914*, in a stuporous condition, and died in spite of the free use of cardiac and respiratory stimulants.

At autopsy a tumor was disclosed as shown in the photograph (Fig. 1). After filling the sella turcica the tumor had evidently found a plane of low resistance laterally, and had invaded the temporal lobe. Although the chiasm was pressed decidedly in the opposite direction, no field defect was found with *5mm* disks at perimetric distance. While it is possible that examination with the test objects of small visual angles might have shown some slight field defect during the last year, it must be noted that a relatively high percentage of acromegalics showing a large sella by X-ray do not show field defects. Evidently in this case an automatic decompression took place laterally at a pressure just short of that necessary to produce a visual defect. Very possibly, however, the same pressure in another individual may have given a marked field defect. Indeed the study of specimens leads one to believe there is almost as much individual variation in tolerance to pressure on the optic nerves as there is in tolerance to the various toxic agents such as tobacco, alcohol, etc.

The following case shows the initial stage of an homonymous hemianopsia due to a primary pituitary struma distending the sella and extending laterally sufficiently to irritate the uncinate gyrus. The tardy appearance of the field defect is again notable.

CASE 2 (Surgical No. 28132). *Hypophyseal struma with hypopituitarism* (adiposo-genital dystrophy). Early homonymous upper quadrantal defects in Stage 1.

July '23, 1911. T. M. E., aged 36. *Complaint:* Uncinate seizures.

Examination on Admission: A fairly typical case of hypopituitarism, with enlarged sella, uncinate fits, diffuse adiposity, hypotrichosis, high carbohydrate tolerance, high blood-pressure, etc.

Eyes: No exophthalmos, diplopia, amaurosis, nor nys-

ILLUSTRATING DRS. WALKER'S AND CUSHING'S ARTICLE ON "DISTORTIONS OF THE VISUAL FIELDS IN CASES OF BRAIN TUMOR."

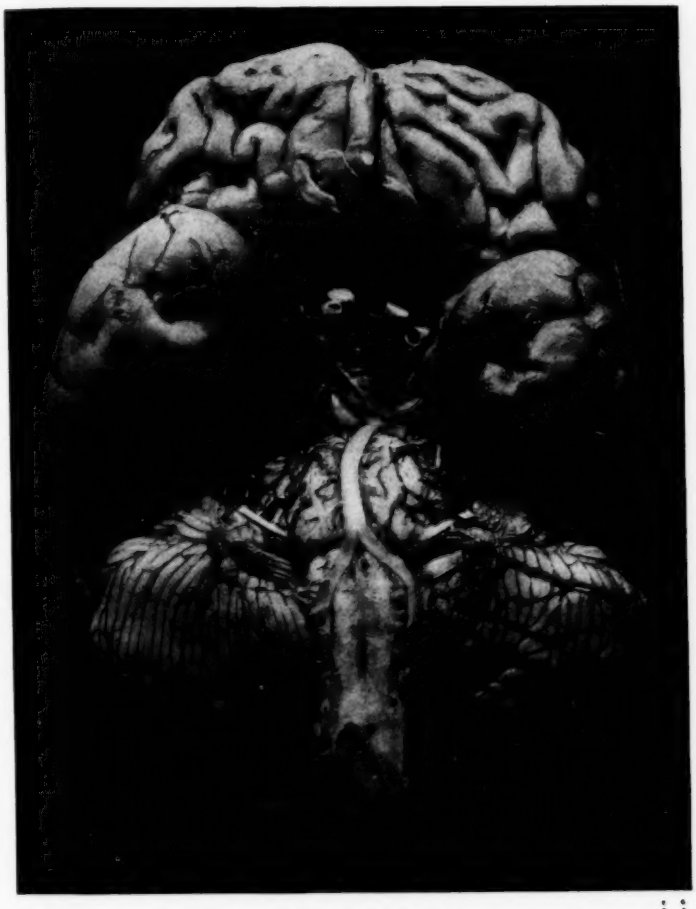

FIG. 1.—Case 1. Showing marked distortion of the chiasmal region by a sellar growth extending laterally without producing a homonymous field defect as might be expected.

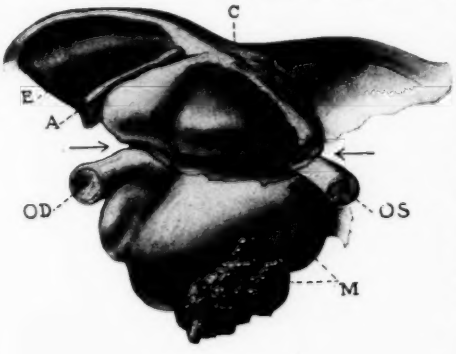

FIG. 13.—Case 5. Showing from in front the chiasmal distortion produced by an hypophyseal adenoma with intracranial extension. M is the intrasellar tumor mass, C the chiasm lying on top of tumor, OS and OD the optic nerves, which have been cut at the level of the arrow by the band of dura which connected the two anterior and widely separated clinoid processes. An artery (A) has also deeply cut the right nerve near the chiasm where it overlies on extension of the tumor (E) out into the right temporal lobe.

tagmus. *Fields* (Fig. 2) were supposed to be normal. V. O. D. 20/20; V. O. S. 20/20.

Glandular treatment, with improvement.

Nov. 6, 1911. Readmission for operation, owing to continued epileptiform seizures. Fields taken by Dr. Blair of Pittsburgh on Oct. 30th, and also those taken at the Johns Hopkins Hospital just before operation, showed a tendency toward a left upper homonymous color notch; but this was not appreciated at the time.

Nov. 9, 1911. Operation: Transphenoidal operation with partial extirpation of large struma.

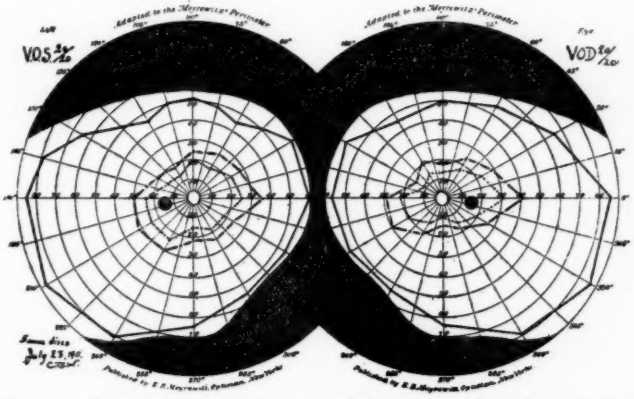

FIG. 2.—Case 2. Fields taken on admission and considered practically normal as far as a diagnostic lead was concerned.

A number of fields taken subsequently by ourselves and by Dr. Blair are practically the same as those before the operation.

Aug. 3, 1912. Patient returned for examination. *Fields* (Fig. 3) show a further advance in the left upper homonymous notch, making the homonymous character of the lesion unmistakable and certifying the insignificant changes of the earlier charts. An homonymous relative scotoma was also present. Fundi and acuity normal.

Sept. 23, 1912. During the past two months periodic lapses of memory have occurred in addition to an aggravation of the uncinate fits of gustatory character.

Oct. 5, 1912. Fields taken on return for examination show a slight decrease of visual acuity to V. O. D. 20/20 and V. O. S. 20/20−, and an increase in homonymous defects.

Oct. 8, 1912. ***Operation*** at Corey Hill Hospital. Second transphenoidal operation. Considerable bleeding at the time of operation but the patient made an uneventful recovery.

April 15, 1913. Further aggravation of memory disturbance and uncinate attacks. Disposition becoming more morose and apprehensive.

April 20, 1913. ***Eyes***: Optic disks show a slight increase in pallor. Otherwise the fundus has not changed in appearance. ***Fields*** (Fig. 4) show slight increase in upper left

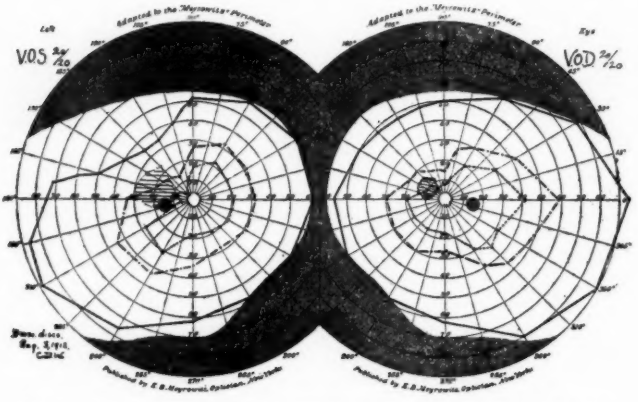

FIG. 3.—Case 2. Early left homonymous defect to color and form, with a relative left homonymous scotoma.

homonymous quadrantal defects: V. O. D. 20/20+; V. O. S. 20/20+.

April 26, 1913. ***Operation***: Third transphenoidal operation. Difficult prolonged operation from which the patient did not rally sufficiently to have further field examination.

Comment. The foregoing observation is of unusual interest from many aspects. In the first place the patient, who for some ten years had consulted many physicians for headaches, adiposity, and impotence, began to have uncinate seizures, the character of which was recognized, but it was not appreciated that they were neighborhood symptoms of an hypophyseal tumor until the X-ray was taken which disclosed a greatly ballooned sella. It is remarkable, therefore, that a primary

hypophyseal struma reaching such a size as to irritate the uncinate gyri should have failed to implicate the optic nerves in the course of its enlargement.

Many fields were taken by ourselves and others, and all were regarded as practically normal, until the last fields here reproduced made a left upper homonymous defect unequivocal. Of course, looking back at previous fields (Fig. 2) in the light of later findings, it was possible to find traces of the condition, which, however, could not have been at the time safely inter-

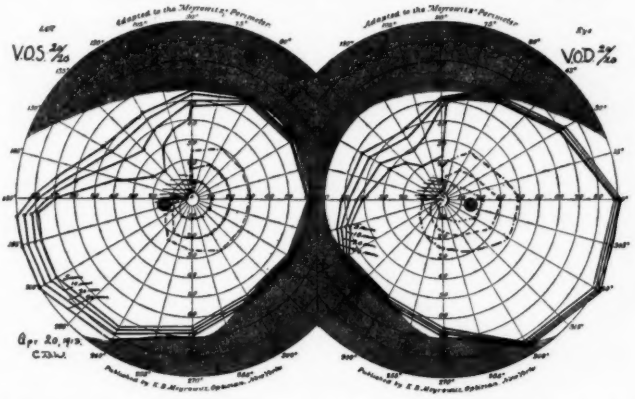

FIG. 4.—Case 2. Further increase in homonymous defect, showing response to large disks in defective region, indicating a state of physiological block and not atrophy.

preted. Here again examination with disks of very small visual angle would doubtless have brought out the real status of affairs much earlier. There are two other instances in the series of cases almost as early as this, but as we pass to the group having a stage of field defect more advanced but still short of complete homonymous hemianopsia, the number increases and the post-operative results have been more satisfactory, possibly because the visual disturbance occurred before the growth was too extensive.

CASE 3 (Surgical No. 1140). *Acromegaly: polyglandular syndrome: diabetes.*

April 11, 1914. Mr. J. B., aged 47. Acromegalic

changes for past ten years, with feeling of "pressure in head." Headache extremely severe last six months. X-ray shows enlarged sella turcica. Glycosuria of 8 per cent., reduced after operation and treatment to 0.3 per cent.

Eyes: Slight exophthalmos. Palpebral fissures wide. Positive v. Graefe sign. Extra ocular movements normal. Convergence good. Joffroy negative. Pupils small, regular, and react to light and accommodation and consensually. *Fundi*. Disks have an injected appearance, but margins are sharp and lamina cribrosa is sharply stippled at the bottom of centrally situated optic cup.

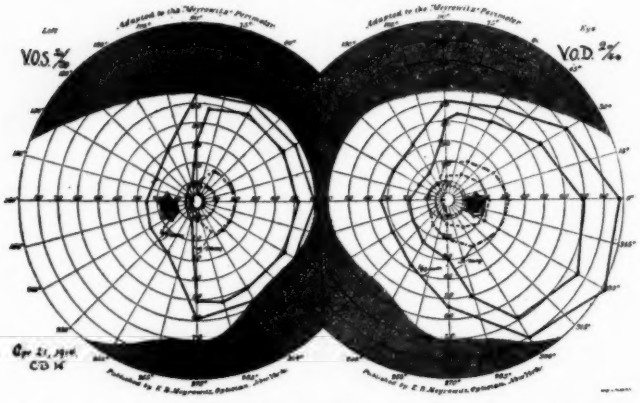

FIG. 5.—Case 3. Fields before operation, illustrating type of uniform shrinkage of homonymous fields.

April 21, 1914. Fields (Fig. 5) show an incomplete left homonymous hemianopsia. V. O. D. 20/40; V. O. S. 20/30. Headache and restlessness prevented prolonged examination.

May 2, 1914. Operation: Transphenoidal operation, second stage. Sellar decompression. Uneventful recovery.

May 7, 1914. Fields (Fig. 6) show a marked improvement in the homonymous defect, and a central vision above normal in each eye: V. O. D. 20/15; V. O. S. 20/15, which has been maintained up to the present time.

Comment. The action of the field defect in this case suggests that the lesion affected the right tract primarily and was possibly pressing from within outward in view of the relatively more advanced damage to crossed fibers representing the left temporal field. The post-operative recovery, very marked

and prompt as it was, substantiated this view, for the tumor must have been resting, in large part at least, on the floor of the sella in order to be released by a midline sellar decompression.

A variation from this type of rather uniform and symmetrical failure of the affected regions, is seen in the following case, where the gourd-shaped configuration so commonly seen in bitemporal cases is found in the homonymous type. Occasionally this conformation of defect is seen in the nasal

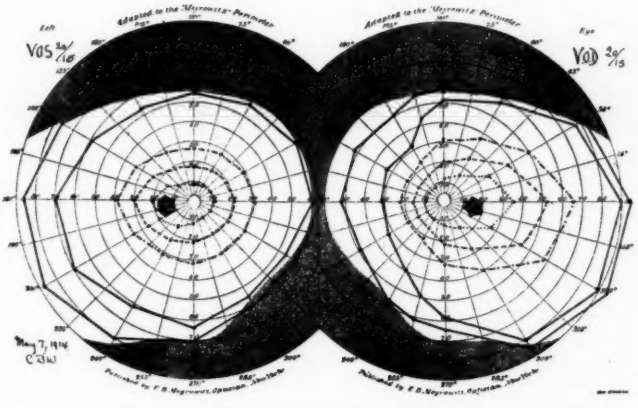

FIG. 6.—Case 3. Fields after operation, showing a type of recovery similar to the type of onset, in its uniformity.

field as well as in the temporal field of the homonymous field defect in this group, but it is more apt to appear in the temporal field only. It would seem to suggest a similar mechanism to that in the bitemporal group.

In the following case—one of typical outspoken acromegaly—the hyperplastic gland had undergone its so-called adenomatous malignant transformation and had presumably broken through its meningeal envelopes to the left of the chiasm.

For a time after the operation the condition in the right eye continued to advance, though there was an immediate improvement in the left. The final rapid restoration did not occur until X-ray therapy was instituted.

CASE 4 (Surgical No. 28552). *Acromegaly. Extrasellar extension of struma. X-ray therapy.*

Oct. 12, 1911. Miss F. J. D., aged 54. Headaches for eight years, boring and occipital in character. No nausea nor vomiting. Vision began to fail about twenty-one years ago, and she has never had the full use of her eyes since. Three years ago her eyesight grew decidedly worse; her appetite became enormous, and her hands and feet began to enlarge. She became somewhat mentally obtuse, and soon began to increase in weight. Failing lateral vision was first noted about one year ago; never diplopia nor

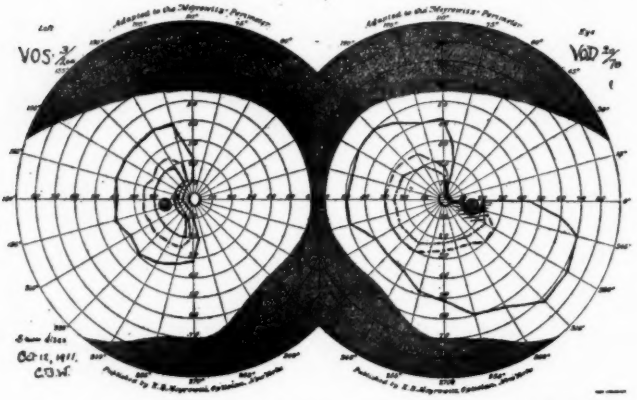

FIG. 7.—Case 4. Fields before operation, showing *gourd-shaped* tendency of temporal defect as in bitemporal hemianopsia.

exophthalmos. Polyuria for the past three or four years; also some epistaxis.

Examination on Admission: A typical case of acromegaly. X-ray shows an enlarged sella turcica.

Eyes: No diplopia, strabismus, nor exophthalmos. Pupils equal; react sluggishly to light but fairly well to accommodation. No nystagmus; movements complete. No v. Graefe. Consensual reaction very poor. *Fundi:* O. D., wide optic cup about 2 D. deep; normal color, perhaps slightly hazy nasally; vessels smaller than normal; lamina cribrosa clear. O. S., optic disk paler than normal, as in primary optic atrophy; lamina cribrosa sharp at bottom of wide optic cup 2 D. deep; fundus otherwise as in O. D. Fields shown in Fig. 7: V. O. D. 20/70; V. O. S. 3/200.

Oct. 13, 1911. Operation: Sellar decompression with

partial removal of fragments of hypophyseal struma. Good recovery, but during the course of a severe cold the visual

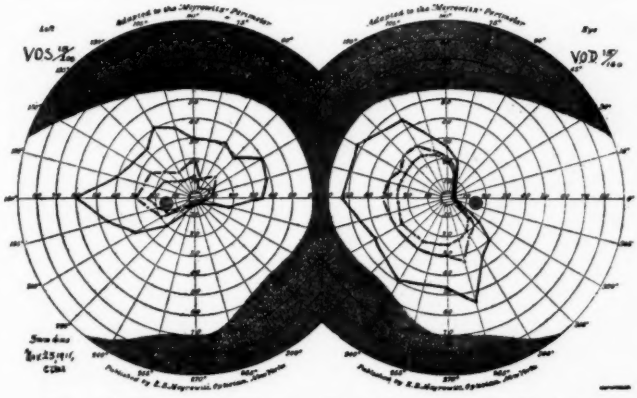

FIG. 8.—Case 4. Showing peculiar post-operative readjustment.

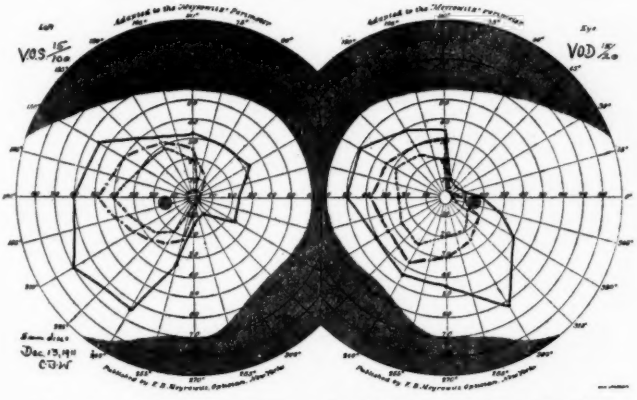

FIG. 9.—Case 4. Improvement in field extent, more marked in the eye most damaged before operation.

acuity sank to almost nil. Subjective improvement following this was fairly rapid, and on *November 23d* the fields shown in Fig. 8 were taken: V. O. D. 15/100; V. O. S. 15/200.

December 13, 1911. Fields shown in Fig. 9: V. O. D. 15/20; V. O. S. 15/100.

Dec. 30, 1911. Fields show further improvement, with one hazy island in temporal field of O. D. (Fig. 10): V. O. D. 20/20; V. O. S. 20/100.

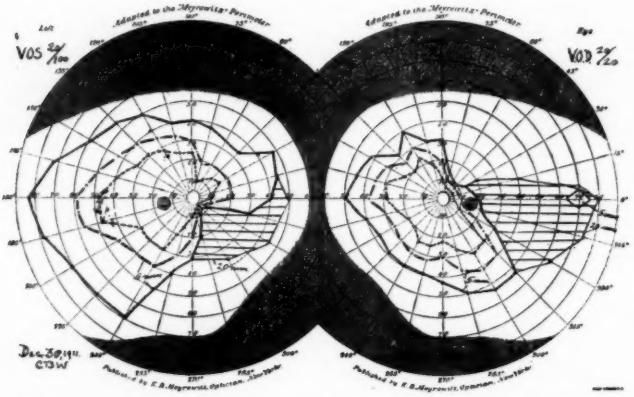

FIG. 10.—Case 4. Further improvement with marked tendency to persistent upper temporal and lower nasal defect. Note appearance of *temporal island* and manner in which larger disks were first used.

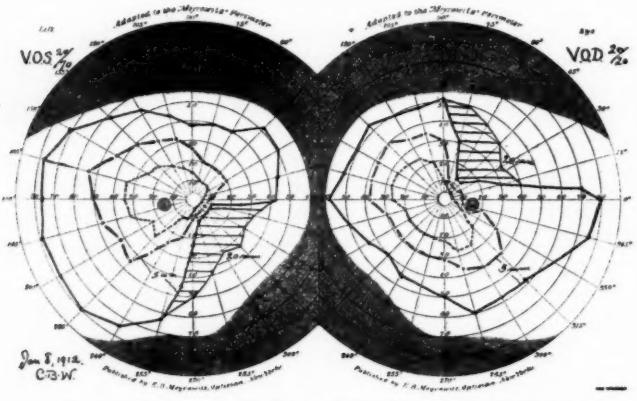

FIG. 11.—Case 4. Final record showing a very satisfactory visual improvement.

Jan. 8, 1912. Fields (Fig. 11) show still further improvement: V. O. D. 20/20; V. O. S. 20/70.

Comment. This patient described the early visual disturbance in a manner to suggest that the field defect was at first bitemporal in tendency but later became more homonymous in character. This is quite possible and indeed the shifting ability of the tumor and field defect in some of these cases is noteworthy in this series of fields. However, the more advanced nasal defect in the left eye would suggest that the tumor had broken through laterally to the chiasm so that pressure came to be predominately against the left tract, particularly the uncrossed bundle. To what extent this pressure was augmented by counter pressure at the foramen or clinoid process is uncertain except as interpreted by a study of the final readjustment in which we find a lower left nasal and an upper right temporal field defect. If this condition represents one of the stages during the onset, it could be accounted for by a pressure beneath the chiasm and anterior tract, and leaning laterally sufficiently to produce the temporal defect and pressing the left nerve against the sharp edge of the foramen above sufficiently to block the dorsal uncrossed bundle to give the lower left nasal defect. We have seen a tendency to this sort of diagonal field defect in other cases of this group, but never in the posterior homonymous group. Such a tendency with dyspituitarism speaks for a tumor extending far enough forward to involve the optic nerves either directly or with the aid of counter pressure in the region of the foramen.

Several of these points as well as previous statements concerning the degree of distortion necessary to produce certain field defects are well illustrated by the following case.

CASE 5 (Surgical No. 4310). *Acromegaly with tumor. Feb. 21, 1916.* Mr. J. H. D., aged 48. For past eight years gradual skeletal overgrowth with increase in weight, about ten pounds a year. Gradual increase of drowsiness and impotence over same period. Without headache, vision began to fail so that the right eye was practically useless four years ago, and the left eye became affected a year and a half later.

Two years ago the fields showed a complete loss of the nasal field of the right eye, with considerable shrinkage of the remaining temporal portion, while the left showed a large cæco-central scotoma and an upper temporal limitation

peripherally. Sella turcica enlarged. V. O. D. 20/100+; V. O. S. 20/30−. Operation was deferred at this time (two years ago), and since then the left eye has grown somewhat worse and the right eye slightly better.

Eyes: No imbalance, no exophthalmos. Right pupil slightly larger than left; both react sluggishly to light and accommodation. *Fundus O. D.* Disk margins sharp. Optic cup shallow. Lamina cribrosa can be made out but rather hazy. Decided pallor of disk, especially temporally, evidently atrophic. Fundus otherwise good condition. *Fundus O. S.* Similar in all respects except the disk, though pale, has much better color than in O. D. Fields (Fig. 12)

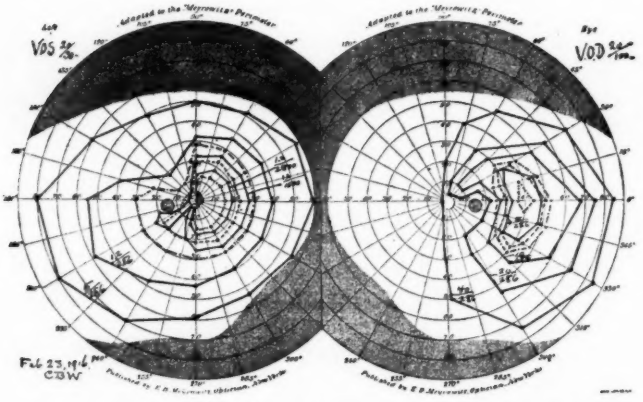

FIG. 12.—Case 5. Fields taken before operation with graduated disks, which give by the quantitative method much additional information.

show a left homonymous hemianopsia incomplete in the left eye with decided central scotoma in the right eye: V. O. D. 20/100 −; V. O. S. 20/30 −.

Mar. 3, 1916. Operation: Transphenoidal. Partial removal of pituitary struma. Poor recovery from the operation. Much respiratory difficulty.

Mar. 5, 1916. Respiratory failure. Death.

From this case a specimen (Fig. 13) was obtained, showing a more marked degree of chiasmal and optic nerve distortion than was expected from the appearance of the fields. Probably the same amount of distortion in another individual would have produced a much greater amount of field damage. Such an individual variation might be due to variation in the firmness of anchorage of the optic

nerves at the foramen and consequent variation in ability to lengthen under tension, as well as to difference in ability to withstand pressure. Here again there is indication of greater damage below in the field of one eye and above in the field of the other eye.

These three cases will suffice to show the characteristics of the stages of homonymous hemianopsia between the onset and the stage of complete hemianopsia. It is notable that there may be a rather uniform general contraction of the homolateral fields or there may be a very symmetrical gourd-shaped stage produced from above or below. One eye usually is more advanced than the other, as in bitemporal cases. As the rind line is reached, central scotomata become bothersome, especially in the more anterior lesions. When a lesion is effective farther back in the tract, as in the following case, the field defect may be more sharply cut in the rind line and central scotomata somewhat more delayed though relatively present.

CASE 6 (Surgical No. 29395). *Hypophysis tumor.*

Mar. 7, 1912. Miss A. G., aged 27. Severe headaches, left temporal and frontal, always associated with nausea and vomiting for past two and one-half years. Six months ago, vision began to fail, until now cannot read. For past three months drowsy and stupid.

Examination on Admission: Fairly typical case of hypopituitarism. X-ray shows obliterated sella.

Eyes: Left pupil twice as large as right. Both react to light, accommodation, and consensually, but left is much more sluggish. Hemiopic pupillary reaction present. Never diplopia. Both eyes prominent, exophthalmos left. No nystagmus. Motion of left eyeball considerably limited.

Fundus O. D. Disk slightly elevated, 0.5 D. nasally. Optic cup not filled but lamina cribrosa cannot be made out. Vascular condition not greatly changed. Fundus otherwise normal.

Fundus O. S. Optic disk pale with sharp margins with optic cup present but indistinct lamina cribrosa, which has a glistening pallor, temporally suggestive of primary optic atrophy. Vascular condition and fundus otherwise as in O. D.

Fields (Fig. 14) show a vertically cleft right homonymous hemianopsia.

Mar. 18, 1912. Operation: Right subtemporal decompression. Lumbar puncture.

April 1, 1912. Fields practically unchanged. Fundi show no measurable swelling; otherwise as before.

April 2, 1912. Operation: Sellar decompression.

April 16, 1912. Fields show no notable change. Fundi show no notable change.

Choked disk is of comparative rarity in the hypophyseal group, due to the fact that the sheath of Schwalbe is blocked off early in the process, so that the usual hydrostatic phenomenon cannot take place, although cerebral pressure, with

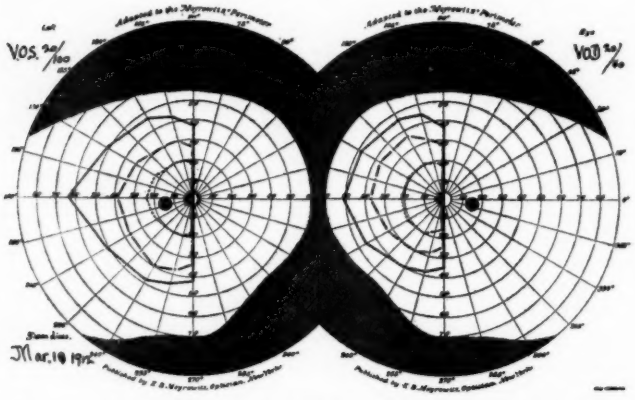

FIG. 14.—Case 6. Fields showing vertically cleft stage and relative central scotomata depressing the central vision,—a condition more common in anterior than in posterior homonymous hemianopsias.

severe headache, nausea, and vomiting, may later take place. When choked disk does occur it is usually moderate and speaks for a growth of considerable size well back in its major extension. However, we have seen a meningitis give the picture of a choked disk of low grade even in a case of bitemporal hemianopsia, so that a hydrostatic pressure in the usual sense may not always be the exciting cause.

In addition to the preceding cases given as examples of the more or less characteristic progress of the defect in the homonymous group, we may give instances where one eye has become blind or nearly so before the other eye shows much or any damage. It may be difficult to classify such cases as bi-

temporal or homonymous in tendency. The deciding factors may be the character of onset in the blind eye obtained from the history or the previous charts, even though poor,—the position in the field of any trace of light perception in case the eye is not absolutely blind, and the character of any minor changes to be found in the good eye. In the following case all of these were available.

CASE 7 (Surgical No. 3781). *Hypophyseal struma with dyspituitarism.*

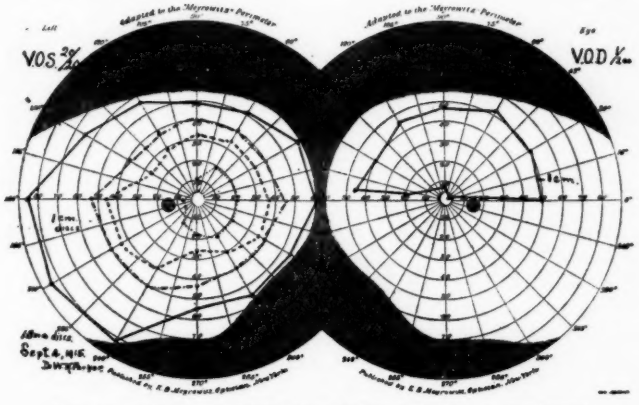

FIG. 15.—Case 7. Fields taken before admission,—equivocal, but with slight suggestion of homonymous tendency.

Oct. 29, 1915. Mr. I. H., aged 33, referred by Dr. Walter R. Parker of Detroit, Mich. An obese individual with characteristic manifestations of dyspituitarism. Frontal and temporal headaches, increasing in severity since onset one year ago. Three months ago discovered that vision in right eye was failing. Fields (Fig. 15) taken by Dr. Parker, Sept. 4, 1915, show a marked defect from below in the right eye, with a beginning upper temporal defect in the left eye. V. O. D. 1/200; V. O. S. 20/20—. The sella turcica was found to be enlarged by X-ray.

Eyes: No exophthalmos, nystagmus, nor muscular imbalance. *Pupils*: Right pupil moderately dilated and shows a very sluggish direct reaction to light. Indirect reaction good. Left pupil gives a good direct reaction, but a poor indirect reaction to light. *Fundi*: Both disks

are pale, but pallor of right disk is decidedly more marked so that a certain amount of atrophy may already have taken place in right optic nerve. The disk margins are sharp and pigmented. The lamina cribrosa is slightly blurred but is nevertheless readily seen. *Fields* (Fig. 16) show an upper temporal defect in the left eye with a central vision of 20/30, while in the right eye there is left only a small uncertain area of light perception in the upper temporal quadrant.

Nov. 6, 1915. Operation: Transphenoidal operation for pituitary struma.

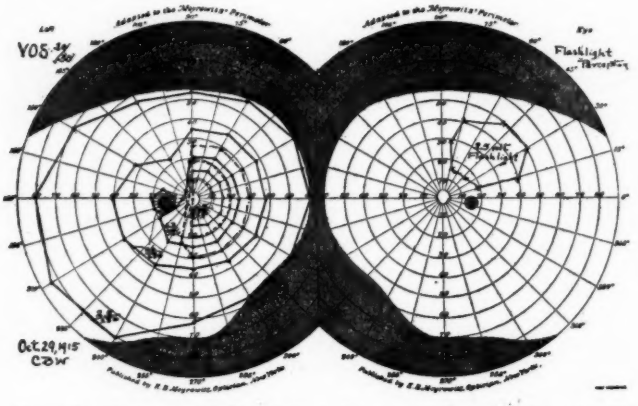

FIG. 16.—Case 7. Field taken before operation, showing definite homonymous tendency, as indicated by residual light perception in upper right temporal field.

Nov. 10, 1915. The return of vision in the right eye was extremely rapid, being easily the record not only for rapidity but also for the quality. The vision had returned to 20/20 in three days after the operation, and to-day, the fourth after the operation, the fields (Fig. 17) show an almost normal outline in the left eye, even to the finest disks, and in the right eye, except for a lower nasal defect, the visual peripheries are practically normal. Remarkably good central vision. V. O. D. 20/20+; V. O. S. 20/15−.

Nov. 15, 1915. Patient discharged in excellent condition.

Jan. 12, 1916. Report of Dr. Parker states that patient is back at work in no way inconvenienced, either by the lesion or by the result of the operation. A very slight lower nasal defect persists in the right field. V. O. D. 20/15+; V. O. S. 20/15+.

June 5, 1917. A communication from Dr. Parker shows that vision has held the same and the fields are normal to the 5*mm* disks.

Comment. The recovery of vision after operation in this case from practically blindness of the right eye to practically normal vision in four days is the most startlingly rapid recovery we have seen, though some others in the bitemporal group have approached quite closely to this record.

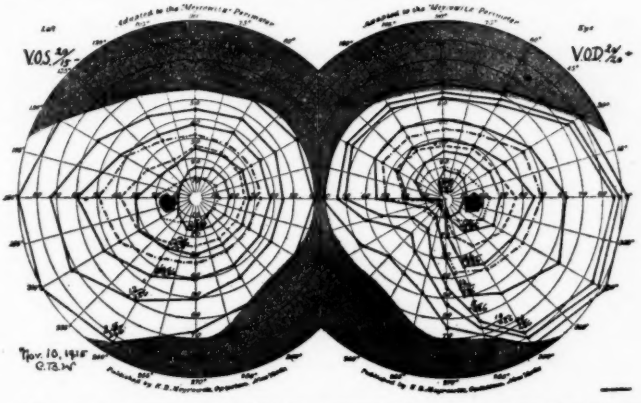

FIG. 17.—Case 7. Astonishing amount of field and central vision recovery in *less than four days* after the operation, establishing in this respect a record visual improvement for all transphenoidal operations done up to the present. Further improvement has been reported by Dr. Parker since patient left the hospital.

The question as to whether to classify this case on admission as bitemporal or homonymous could perhaps not be answered from the field taken previously (Fig. 15), though the nasal defect of the right eye seemed slightly more advanced than the temporal, suggesting a homonymous defect in tendency. The field taken on admission (Fig. 16), however, decided the question, since it was found that the small amount of light perception remaining in the right eye was located in the upper temporal portion of the field, and the manner in which the field later recovered left no room for doubt.

It is interesting to conjecture about the mechanism of the field defect in this case. The tumor without doubt, after

filling all the available sellar space and producing sellar enlargement by pressure, broke through to the right so that the right optic nerve was forced upward against the rather sharp edge of the foramen or the rough dural interclinoid margin. This concentrated counter pressure being more effective than the rather diffuse pressure from the tumor resulted in the more marked field defect from below. The transphenoidal decompression allowed the tumor to settle downward and the vision was quickly regained because of the release from acute strangulating pressure on the right optic nerve. Moreover, the duration of this acute pressure was short,—three months, which is a condition always favorable to a rapid recovery since atrophy does not become marked until six months or more.

Here again we see the tendency to an upper temporal and a lower nasal homonymous defect as in the previous case. Whether or not there is some rotation of the nerve as the chiasm is tilted upward on one side, to aid in producing such a result, is difficult to state though it would seem quite possible.

In addition to the foregoing rather typical instances of field defects in the homonymous group, we have those cases showing central scotomata sometimes of large size but without marked peripheral defect. When such a scotoma is present in one eye only and a temporal hemianopsia present in the other, as in the following case, we have a condition that may readily develop into a bitemporal or a homonymous hemianopsia.

CASE 8 (Surgical No. 4409). *Pituitary group. Dyspituitarism with struma.*

Mar. 12, 1916. A. O., aged 34. Severe frontal headaches every month or so for years, growing markedly worse in last three years and tending toward left side of head. Some nausea with these headaches in last six months. Loss of left lateral vision first noted 2½ years ago. Also loss of libido. Moderate polydipsia and polyuria. Inconstant diplopia for last year.

Eyes. Left pupil about 1½*mm* larger than right; both regular in outline but left rigid to light and accommodation, direct and indirect, while right is active to same. Partial paralysis of left internal rectus giving slight external strabismus. Slight ptosis of left lid. No exophthalmos or nystagmus. *Fundi* normal except for slight increase of

disk pallor. Pallor of both disks of same grade. Lamina cribrosa sharp. Optic cup well formed.

Fields (Fig. 18) show a temporal hemianopsia in the right eye, and a large central scotoma in the left eye. V. O. D. 20/30; V. O. S. ½/200.

Mar. 20, 1916. Operation. Transphenoidal procedure with partial removal of soft struma. Recovery from operation, followed by disappearance of all subjective discomfort.

April 10, 1916. Fields (Fig. 19) show considerable improvement: V. O. D. 20/20; V. O. S. 10/200.

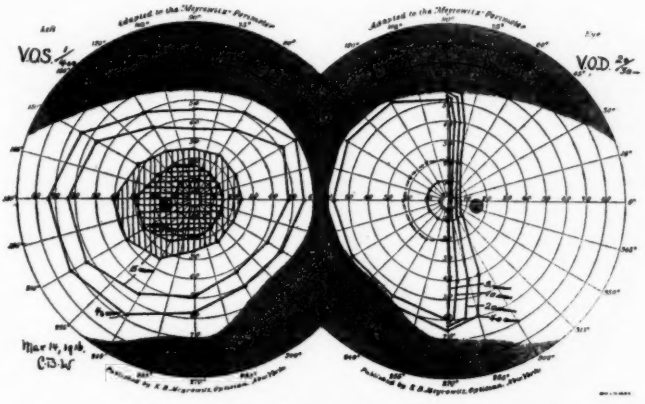

FIG. 18.—Case 8. Fields taken before operation, showing central scotoma in left eye and hemianopsia in right eye associated in such a way that further visual failure in the right eye may throw the case into either the homonymous or bitemporal group.

Comment. While the more advanced defect in the right temporal field suggests a mesially disposed pressure which would result finally in a bitemporal hemianopsia, there is also evidence in the tendency of the scotoma to remain slightly more extensive in the lower nasal field of the left eye after operation that a homonymous tendency is present. The presence of such scotomata as this speaks more often for a tumor than a struma, in which case lack of symmetry might be expected, resulting in a homonymous type of field. It is difficult, if not impossible, to explain a scotoma of this character on a mechanical basis alone, so that we find ourselves

reduced to the rather unsatisfactory explanations based on hypersensitiveness of the papillo-macular bundles or possible toxic actions.

GENERAL COMMENT.

Certain cases in this group have shown remarkable improvement in vision after the transphenoidal operation. Most striking of these is Case 7 where a blind eye recovered normal

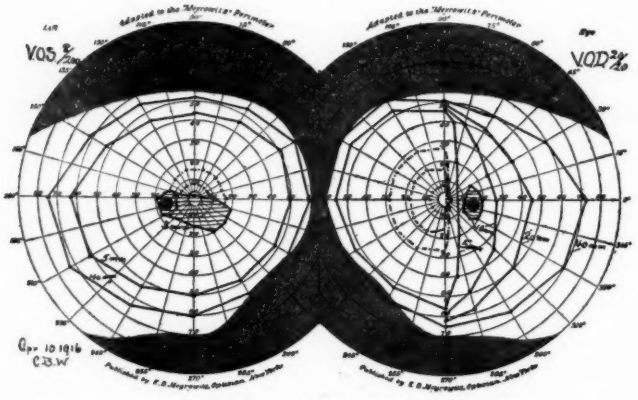

FIG. 19.—Case 8. Fields afer operation, showing visual improvement. Slight evidence of homonymous tendency.

vision in three or four days with an almost complete field recovery. Case 4 also shows a slower but very satisfactory recovery of vision. But altogether, from an operative standpoint, the results are not so satisfactory in this group as in the bitemporal group as regards improvement in vision. This of course is accounted for by the fact that most of the lesions in the homonymous group are tumors rather than strumas and have a tendency to grow more wilfully and unsymmetrically.

It is notable that the extent and size of the tumors may not give a very exact idea of the amount of chiasmal distortion present. Thus in Case 1 a marked distortion of the chiasm failed to give any decided field defect such as might be expected. Also in Case 5, considering the amount of distortion and compression shown in Fig. 13, a considerably greater

field defect would not have been surprising. Possibly individual variation, not only in the response of the optic fibers themselves to insult but also in the firmness with which the optic nerves are held in their foramina, may have a decided influence on the resulting field defect.

While the operative results in the homonymous group of pituitary growths are not so uniformly successful as in the bitemporal group, because of the difference in the nature of the growths, nevertheless surgical interference has been amply justified by brilliant results in certain cases and in checking the failure of vision in others.

HEADACHE AS THE RESULT OF EYESTRAIN.

By JOHN DUNN, M.D., Richmond, Va.

YEARS ago the problem headache was led to the door of Ophthalmology and left there. Certain relations between the use of the eyes and the production of headache were noted, and upon these observations the art of applied refraction, including the determination and suggestions for treatment of muscular imbalance, was built up. So far as I know, however, ophthalmology has made no effort to answer the question: How does the correction of refractive errors relieve headache? Or, a step further back, the other question: What is headache?

To say that the use of glasses lessens the strain on the ciliary muscles and so relieves or prevents headache—even though true—is no answer at all. How does the use of the ciliary muscle produce headache any more than use of the deltoid or the peronei? If use, or over-use of the ciliary muscle produces headache, why does it not always do so? In the scheme of the human economy, nature plays no favorites. Mrs. Brown is forty and is both farsighted and slightly astigmatic; has used her eyes for close work as much as she chose to; has never worn glasses, and has never had a headache in her life. Mrs. Smith, aged forty, has an equal degree of farsightedness and astigmatism; has no imbalance of the external ocular muscles, and yet for years the slightest prolonged use of her eyes for close work, unless she was wearing proper glasses, has in a large proportion of the times resulted in headache. Mrs. Jones, aged forty, with the same external muscular conditions, and the same refractive errors as Mrs. Smith and Mrs. Brown, can use her eyes neither with nor without glasses that the result is not invariably headache. Baldly stated there is the problem.

To say that Mrs. Smith has a general condition which lessens the tone of the ciliary muscle, and that Mrs. Jones has some physical trouble which still further increases the difficulty of response on the part of the ciliary muscle to retinal-born impressions and demands, is to bring us a doubtful step nearer, but such an answer still begs the question: Why should use of the ciliary muscle produce headache and not use of the sartorius do the same? What relation has use of the ciliary muscle to the production of headache? Why not jaw-ache, or toothache, or earache? Why headache? The ciliary muscle is a structure outside of the skull. Headache is a disturbance which resides inside the skull.

Again, take the case of Mrs. L., aged 36, who has oblique hyperopic astigmatism of 1½ dioptries in each eye. To go without correcting glasses for even a short while results in headache, vertigo, and nausea. Wearing her glasses she is able fairly comfortably to do a full day's general work. Exercise of the muscles of her arms over the wash-tub produces with her neither headache, vertigo, nor nausea. Exercise of her accommodation, with the refractive errors uncorrected, produces all three. It is plain that the headache, vertigo, and nausea are not simply the result of a continual call on the brain cells for the origination and continued co-ordination of muscular impulses—in other words, of sustained attention. If so, the work over the wash-tub would cause them. The vertigo and nausea are capable of explanation as the result of excessive irregular, perhaps spasmodic, contractions of the ciliary muscle. The impulses thus originated reach the medulla and overflow into the vagus center (nausea and vomiting) and into the vestibular centers (vertigo). That this is probably the explanation of ocular vertigo and nausea, can be inferred from the ease with which vertigo and nausea can be aroused by disturbance of the fluid in one or more of the semicircular canals. Here the nerve impulses from the canals after reaching the triangular nucleus in the medulla over-stimulate the adjacent vagus centers and may produce pallor of the skin, sweating, nausea and even vomiting—*but not headache*. What is it that causes the headache? Again, take another of the symptoms found so frequently accompanying headache secondary to eyestrain. I have reference to what the patient

terms "nervousness." "Doctor, when I have one of my headaches, I get so nervous I could jump out of my skin." A temporary and transient symptom this is with some cases. With others, especially where there is a rather high degree of compound hypermetropic astigmatism with external muscular imbalance, the "nervousness" becomes, as the years go by, an almost constant symptom and the predominant one in the patient's mind and in that of the minds of those with whom he or she is surrounded. To what is this nervousness due? As I see it, there is only one satisfactory explanation, and that is that it represents a reflex instability of the physiological intracranial pressure, the predominating phases of which is a pressure above normal. The extreme phases of this nervousness are to be found in certain cases of acute intracranial inflammation and injuries.

What is headache? A pain in the head? That will not do. We are after a definition which explains, not one that defines. We are chasing game in the fields of medicine, not in the pages of a dictionary. Why should the head ache after use of the eyes? Because the eye is innervated by nerves which have emerged from the brain? So are muscles of the auditory drum membrane and yet response of the tensor tympani to sound waves does not produce headache. What brings about the headache? What are the actual intracranial conditions which cause the headache? How do these conditions come into existence?—in some cases so easily, in others with such relative difficulty.

Such is the problem before us and I have presented it in the foregoing form that I might, as far as I could, divest it of all its superfluities and so prepare the question for the best answer I can give.

Headaches, inclusive of those due to injuries and to tumors, *are a manifestation of increased intracranial tension.*

Let us examine the headaches in a case of brain abscess and in one of brain trauma.

> Jno. K., aged 26, showed symptoms which suggested the forming of a right-sided cerebellar abscess. Three or four days later he became wildly delirious, his pulse dropped to about 30 to the minute, and he began to scream repeatedly at the top of his voice as if in the most intense agony. The

bone was removed over the right half of the cerebellum. A deep-seated abscess was found and evacuated. Immediately following the opening of the abscess, the pulse jumped to 140 to the minute. Within a very short while the pulse was regular and 84 to the minute; the patient was perfectly conscious and the pain in his head had disappeared. The severe headache was not due to the presence of the infectious material within the cerebellar substance, for although the abscess was opened and much thin purulent matter ran out, the cerebellar substance immediately closed in upon the abscess cavity, obliterating it and the tract by which the pus escaped. The infection remained, as was proven later by a reaccumulation of pus, pressure upon the respiratory center, and death—*but no further headache.* The headache was not one due to meningeal inflammation, because of its disappearance practically immediately after the opening of the cranial cavity. It may be safely said that the headache was a manifestation of an acute rise of intracranial tension, and in this case one due to blockage within the region of the lower water-bed, probably of the iter itself.

Again take the case of Mr. L., who fell from the roof of a shed, striking the right temporal region against a pile of bricks. The next day he was wildly delirious, screaming with pain and unconscious. The removal of the right temporal plate and ligation of a branch of the middle meningeal artery and evacuation of a clot resulted in the restoration of consciousness and disappearance of both delirium and headache in a few hours. It was not the injury to the skull cap which induced the symptoms nor the injury to the dura, nor the rupture of the middle meningeal artery. It was the increasing intracranial tension, for none of the symptoms followed immediately upon the receipt of the injury.

Similar instances of headache following upon acute rise of intracranial tension could be multiplied indefinitely. The above two have been mentioned here to show that the factor in the production of the headache in such cases is not the cerebritis; nor injury to the dura or to the bony cranium, or to the meninges, but to a rise in the intracranial tension, a subject about which a great deal has been written. The medical literature of this especial subject has to deal with that type of intracranial tension which accompanies brain tumors, abscess, meningitis, and cranial injuries. Relatively little has been said about what may be called *physiological increased*

intracranial tension, its cause, and method of production and regulation.

What constitutes a normal intracranial pressure? The intracranial pressure is normal so long as it is equally distributed over all parts of the brain surface and admits of an intracranial circulation of the blood just sufficient for the full needs of brain metabolism.

What is cerebrospinal fluid? It is a "colorless, limpid fluid of low specific gravity, among whose grosser chemical characters are its low protein content and the presence of a definite reducing body known to be glucose." Wegefarth and Weed (*Journal Med. Research*, vol. xxxi., 1914–15). It is elaborated chiefly by the choroid plexuses in the lateral ventricles, and is found intracranially in the ventricles, in the water-bed of the posterior fossa, and generally in the meshes of the pia-arachnoid.

How does it escape from the interior of the cranium? Key and Retzius (1876) claimed this occurred through the Pacchionian bodies into the longitudinal sinus.

Dandy and Blackfan (*Am. Journal of Diseases of Children*, vol. viii., 1914) as the result of their experiments came to the conclusion that it found its way out by "the exposed capillaries of the pia-arachnoid" and so into the veins.

Weed (*Journal of American Research*, vol. xxxi., 1914–15) says "the anatomists (J. Mestrezat, W) are agreed that the arachnoidea is non-vascular. Weed further says he has never observed any evidence of a capillary bed in the pia. Weed came to the conclusion that the absorption was through the arachnoid villi wherever found. The distribution of these villi in order of frequency is—superior longitudinal sinus, transverse sinus, cavernous sinus, and venæ meningiæ mediæ. That these villi communicate with the fixed venous channels does away with the variations in pressure which occur in the free veins, a precaution which only serves to accentuate the extreme delicacy of all the brain processes. This anatomical provision lends weight to the correctness of Weed's observation. Some of the cerebrospinal fluid escapes from the spinal canal. Weed's experiments seem to show that it is relatively slight in amount. By what passages this escape is made is unknown. The problem of the secretion of the cerebrospinal

fluid, however, is not solved by having discovered its source and its channels of escape. Nor do the results of analyses answer all the questions.

Weed (vol. *cit.*) says: "The mechanism of the cerebral pressure relations is such that the cerebrospinal fluid pressure is continually being reduced to that of the venous circulation and, in consequence, there must be a constant absorption of the fluid to compensate for the constant secretion." What is it that regulates both absorption and secretion? The cerebrospinal fluid makes the exact intracranial pressure adjustment possible, but there must be something which determines the volume of output from the choroid plexuses and the correspondingly controlled passage into the fixed veins. What is it that regulates the degree of permeability of the fluid itself? These things are not merely mechanical. They must be presided over by an extremely delicate nervous mechanism. The slightest undue pressure upon the cortical cells calls for instant adjustment. The position of the hypophysis and the fact that its posterior lobe secretion passes into the third ventricle suggests that its rôle is in part to regulate the intracranial pressure. Bell says that even though "the secretion from the pars nervosa does pass into the cerebrospinal fluid, as has been asserted, there is not the slightest evidence that this is essential, beneficial, or even the normal method by which the internal secretion is taken up by the animal economy." (*J. A. M. A.*, p. 1698, 1917.) This may and probably does apply to the activities of the anterior lobe. Indeed, we have reason to infer this from consideration of the other glands of internal secretion. The output of no other of these glands is poured into a space similar to that into which the posterior lobe empties its secretion. Why has the posterior lobe its peculiar channel? The cranium, the eyeball, and internal ear have each a water system, the prime object of which would seem to be to make possible an even pressure upon the cortical cells of the first and upon optic and auditory and vestibular terminals of the other two. If we look at the mechanical arrangement of the water system of internal ear, we see that it is so constructed that under normal conditions the pressure upon the auditory and vestibular terminals therein varies with the intracranial pressure. At first glance it

would seem that no such provision has been made for the eyeball. Wegefarth's (*J. of M. Research*, vol. xxxi.) experiments, however, would seem to prove that "by varying the pressure relations existing in the cranium and in the eye" there is "a direct connection of the two fluid spaces, at least for true solutions." It would seem further, however, that under normal conditions the water system of the eye is sufficient to regulate the pressure upon the optic terminals without the assistance of the intracranial system *save for one thing*, and that is, it cannot furnish to the aqueous that something whose presence we may infer is necessary to regulate the permeability of the aqueous after its secretion. There must be some provision for controlling both the output and the escape of the cerebral fluid. The distal situation of the arachnoid villi and the driving force of the intracranial circulatory impulses are some of the mechanical factors; but we are dealing with a problem different from a pumping station and hydraulic outlets. The intracranial pressure has to be rapidly regulated within almost infinitesimal limits to meet the normal requirements of the cortical cells. There must be some reflex arrangement whereby the permeability of the cerebrospinal fluid is made to vary. It is difficult to imagine a way by which this can be done save by the addition to the output of the choroid plexuses of a substance to accomplish this end. It has been proven that the arachnoid villi are permeable to aqueous solutions, just as they are permeable to the cerebral fluid itself. This merely shows that the arachnoid villi would mechanically admit the passage through them of a cerebrospinal fluid deprived of its regulative agent and that the purely mechanical part of the intracranial water-way is sufficient to prevent a very high intracranial tension. It by no means proves that a purely watery cerebral-spinal fluid is sufficient for the normal processes of the cerebral tissue. Indeed, there are reasons for believing it is altogether insufficient. The position of the hypophysis and the fact that its posterior lobe secretion finds its way into the third ventricle and is mixed with the output of the choroid plexuses is highly suggestive that this secretion is the substance which has an influence upon the permeability of the cerebrospinal fluid. Deprive this fluid of the posterior lobe secretion and the brain processes go on without its regula-

tive action, but there appear in the course of time many disturbances of bodily growth; these latter, I believe, are to be attributed in part to an imperfectly regulated intracranial pressure. That the choroid plexuses will continue to secrete in the presence of a non-functionating posterior lobe seems to have been proved. It has equally been proved that the bodily processes do not progress normally under the same conditions. It seems more than probable that the activity of the posterior lobe responds reflexly to the general intracranial pressure, *i. e.* of the pressure upon the cortical cells. *It is probable also, that continued higher than normal intracranial pressure does not mean a continuous hypersecretion on the part of the posterior lobe. Functional reflex exhaustion sets in relatively early, and the result is reflex dyspituitarism,* a provision which does away with what would be the necessary result—a continuous hypertension. It is here contended that the symptomatic manifestation of temporary high tension which results from reflex dyspituitarism is headache. It seems further not unfair to infer that the hypophysis responds reflexly not only to changes in pressure upon the cortical cells, but also upon the retinal ganglion cells, a part of the brain substance also subject to variations in pressure.

What part does the ciliary muscle play in the production of a functional disarrangement of the hypophysis? When we compare the eye with the brain, what do we find? Both have an envelope within which the maintenance of a normal tension is one of the requisites for the perfect functioning of its contained nervous structures. The elaborate precautions taken by nature to secure a tension which normally varies within almost inconceivably narrow limits is perhaps exceeded nowhere in the human economy save in the case of the auditory and vestibular terminals to the inner ear. The exquisite delicacy of the vestibular apparatus is probably due to the fact that the instant knowledge of the exact position of our head, without which vertigo ensues, is even more important than sight.

Wegefarth (*Journal of Medical Research*, vol. xxxi., 1914–15) has compared in detail the water systems of the cranium and of the eye. He compares the choroid plexuses and the ciliary glands. He calls attention to the epidermal lining of the

ventricles and of the postiridial chambers and to the mesothelial covering of the pia-arachnoid spaces and of the anterior chamber. He compares the arachnoid villi and their relation to the fixed veins with the "pectinate" villi in their relation to the canal of Schlemm. He calls further attention to the fact that "some of the aqueous finds its way backward between the hyaloid and the inner surface of the retina, making its way about the retinal neurons and axons," observations which have an important significance in the physiology of the retina, as well as of the aqueous." It seems not unlikely that the presence of the aqueous, small in amount though it is, between the hyaloid and retina has for one of its purposes the regulation of the pressure on the retinal ganglia in the act of accommodation. The normal variations of pressure on the cerebral cortical ganglia are the result of the activities of the intracranial circulation and the cerebral fluid elaborating organs. On the other hand, the variations of pressure upon the retinal ganglia are not only subject to the normal changes in the intraocular circulation and the amount of aqueous, but to the relatively more violent activities of the ciliary muscle, whose responses to the higher centers for accurate adjustment of the details of the retinal images are often impaired by refractive errors and by lack of tone due to abnormal conditions of the blood circulating through it. Any over or under activity of the ciliary muscle creates a response in the activity of the aqueous secreting glands and results in disturbance of the normal state of the intraocular tension. That in eyes where the excretory system for any reason is imperfect, whether congenitally or from acquired conditions which will ultimately result in glaucoma, strain upon the ciliary muscle from overuse of the eyes in close work results in transient attacks of higher than normal tension, in some cases measurably so. That a relatively higher than normal pressure is exerted upon the retinal ganglion cells in the ordinary case of eyestrain, I think unquestionable, even though the instruments at our command are not delicate enough to measure this rise. In considering the effect of eyestrain, we must bear in mind that we have to deal not only with an over-exerted ciliary muscle but with an ocular water system whose activities are in a measure correlated with those of the ciliary muscle. The

lessening of the activities of this latter as we pass through the various stages of life and the intraocular compensations therefor form an interesting chapter in the history of the eye. In considering intraocular tension, we must bear in mind that we are not dealing with a problem as mechanical as the flow of water through a trough or with a purely capillary action like the absorption of water into a wick, even though these same physical laws play their part. Although the ciliary activities are responded to in the regulation of the intraocular system, they are not necessary to its continuance, as we can see in cases of paralysis of the ciliary muscle. Nor is the response of the ciliary glands merely a transudation which keeps pace with the escape of the aqueous from the eyeball. This is demonstrated in cases of glaucoma. Under normal conditions the regulation of the intraocular tension is under control of a set of complex nervous reflexes. In all likelihood these closely resemble those which regulate the intracranial tension, if indeed they are not part of the same system; and just as in the case of the brain, the normal activities of the choroid plexuses and posterior lobe of the hypophysis vary with the pressure upon the cortical cells, so in the eye the normal activities of the ciliary glands vary with the pressure upon the retinal ganglia.

The eyeball does not seem to provide an agent capable of varying the permeability of the aqueous. I believe, however, that such an agent is necessary to the perfect functioning of the intraocular water system. Less so, however, than the cerebral system perhaps because of the presence of the ciliary muscle whose activities by varying the intraocular pressure in a way unknown in the intracranial system serve as a substitute. That, however, the ciliary muscle is a complete substitute I think most improbable. Wegefarth, as quoted above, says it has been proved possible by varying the pressure relations existing in the cranium and in the eye to demonstrate the direct connection of the two fluid spaces, at least for true solutions. If this be so, then the eye has a means of obtaining from the cerebral fluid a sufficient quantity of posterior lobe secretion to meet the necessities of the aqueous.

If we admit the above facts and contentions, then we can see that eyestrain which results in disturbance of the normal

pressure upon the retinal ganglion cells causes reflexly a disturbance of the secreting activities of the posterior lobe of the hypophysis. If this continues long enough, reflex temporary exhaustion of the posterior lobe activities sets in and with it higher than normal intracranial pressure—the clinical symptom of which is headache. The frequent association of eyeache after ciliary strain and headache must not be overlooked. Both have the same explanation—hypertension.

Let us examine the symptoms in a few cases of headache.

Mrs. J., aged 47, began to have headaches when she was about thirteen years of age. She has suffered with headaches ever since and they are always worse at time of menstrual flow. Her refractive error is less than one dioptric of farsightedness. There is no astigmatism and no external muscular imbalance. While the use of glasses may have somewhat lessened at times the severity of the headaches, their use did not prevent and seemed in no way to be able to lessen the severity of those of monthly recurrence. In this case the headaches for a short time preceded the full establishment of womanhood and recur with each act of menstruation to the time of menopause. They occur frequently between the menstrual periods and seem then to be brought on by constipation, digestive upsets, physical exhaustion, or strain upon the emotions. The fact, however, that is uppermost in the woman's mind as the result of years of suffering is that the headaches always are more severe at the time of menstruation.

Miss M. B., aged 30, refractive errors none, external muscular conditions normal, a shrivelled, dried-up old woman, "a martyr" her friends all say "to headache." She has never menstruated.

Miss A. J., aged 26, suffers intensely with recurrent headaches and at the menstrual period she is unable to read save with pain; her eyes ache and the sunlight is unbearable. Her refraction and the external ocular conditions are normal.

Miss B., aged 18. Severe recurrent headaches, which are associated with photophobia, pain in the eyeballs and inability to use her eyes, always worse at the time of her menstrual flow, which is accompanied by abdominal pains and pains in the back and nervous symptoms. Refractive and muscular conditions normal.

But why multiply examples? Thousands of similar cases visit the ophthalmologists' offices every month of the year. Question these cases and they tell you of "painful or irregular menstruation," of suppression or absence or irregularity of the menstrual flow, and practically always that their headaches are worse at the time of the menstrual periods. Question their physicians and you get the stories of ante- or retro-flexion, of stenosis, of ovarian cysts, of mal-developments and all the other pictures in the gynecologist's text-books. The result is that ophthalmologists are forced to the belief that there is more than a fancied connection between disturbed ovarian activity and headache. What is it?

None of these cases are as simple as they would appear at first glance. Besides the ovarian abnormalities there will usually be found a history of constipation, and often symptoms which are referable to some chronic focus of infection. How far disturbances of the ovarian secretion has an influence on the activities of the hypophysis and on which lobe cannot be accurately stated. That there is an interrelation of activities is most probable. The disturbance of this interrelationship may have effect in the functioning of the hypophysis in so far as it regulates the intracranial tension and in so far as its influence as explained above produces headache. The concomitant diseases, constipation, digestive disturbances, uterine abnormalities, foci of infection wherever situated, etc., have their effect indirectly upon the ciliary muscle, through changes in the composition of the blood whereby the ciliary effort in accommodation is increased and the difficulties of maintaining normal intraocular conditions are also correspondingly increased. In a similar manner, too, are to be explained the headaches of fever. Every oculist has wondered that so many young people with relatively small errors of refraction "have so much trouble with their eyes," have headache so often following their use and seem to need the assistance of a weak glass. The explanation of this with many cases is that they are suffering from chronic infection of some portion of the exposed lymphatics of the upper air tract, especially of the faucial and post-nasal regions.

Thus we have an explanation why Mrs. Brown needs no glasses, why for Mrs. Smith the use of glasses prevents

headache, and why Mrs. Jones can get little or no relief from their use. In one case, nothing interferes with the normal functioning of the ciliary muscle. There is no disturbance of the intraocular tension and hence no abnormal pressure on the retinal cells. In the second and third cases in different degrees, there are, as the result of blood changes or intraocular abnormalities of development, difficulties in the way of the ciliary muscle continually performing its task without there resulting indirectly undue pressure on the retinal cells, the reflex from which is disturbance of the hypophyseal secretion and, as explained above, headache. We must bear in mind that the retinal cells are as sensitive to changes of pressure as are the cells of the brain cortex and any variation from the normal pressure upon them registers a call for recognition. In the case of the cortical cells, this is taken note of by the intracranial water system. In the case of the retinal cells, there is first an effort of the intraocular water system to give the relief, but where the disturbance is the result of a continuing muscular effort (the ciliary) which is insufficient for the demands made upon it, and where these efforts themselves bring about difficulties in the normal manufacture and escape of the intraocular fluids, there result, on the part of the eye, "pain in the ball," "light shyness," and reflexly, as described above, headache.

In considering headaches, the result of errors of refraction, we must remember that these latter are in any given case constantly present, and that the eye is during the waking hours never altogether free from the "strain" they impose. It is this unremitting over-effort, however varying and slight it may be, that eventually brings about unstable intraocular tension and then reflexly unstable intracranial pressure, which so readily, when the eyes are called upon for excessive effort, result in headache.

This paper would be incomplete without some short comment on a class of cases which has been always, and is to-day, the despair of the ophthalmologists.

Mrs. L. B., aged 41, may be taken for an example. She has had headaches ever since she could remember. It is a headache from which partial surcease has been obtained

for only very short intervals. She has consulted many doctors and submitted with hope and patience to the wearing of many strange medical garments. She has consulted the neurologist, the abdominalist, the internist, the dentist, the surgeon, and the oculist; she has wandered along all the devious paths which lead off from the straight road of medicine, and the headaches continue. Every medical man she has consulted has felt that he has met with something different from the more or less transient headaches with which he has to deal every day, and he undertakes to treat the local trouble, whatever it may be, with a feeling that its relief will in no wise lessen the headache. What is the matter with Mrs. B.? She has what may be termed a "wet" brain, the result of an unrelieved, constant, though varying, high intracranial tension. If I may borrow from ophthalmology a term, I should say Mrs. B. has cerebral glaucoma. The only treatment for them is decompression, but in advising it it should be borne in mind that the operative measures resorted to in the treatment of ocular glaucoma never leave an eye in its first perfect condition and that all such measures are to a great degree only palliative. They, however, often accomplish a great good, and I believe the same may be said of the decompressions done for headache in the cases of "wet" brain.

The ease with which "eyestrain" results in a physiological high pressure on the retinal terminals varies with the development of the eyeball. Where the eyeball is small and the lens is large, and where the spaces of Fontana are not amply developed, it will be seen at once that relatively little cilio-crystalline activity will result in a derangement of the physiological intraocular pressure, the pressure on the retinal element will be increased. Reflexly the intracranial pressure will increase. Headache results and with it enforced rest of the cilio-crystalline system. It looks as though the physiological headache had for one of its objects to bring about this result.

The following two observations are of interest here: "Doctor, in the mornings my head aches terribly and I void frequently large quantities of perfectly clear urine. In the afternoon my urine is normal in color, there is very little of it, and my head ceases aching." The patient was forty-odd years of age; his systolic blood pressure 210; advanced nephritis. Mr. L., aged 50, has noticed from time to time

since he was 20 that his headaches would be accompanied by the excretion of large quantities of "urine as clear as water." This "urinary irritation" would cease with the disappearance of the headache. Furthermore he has learned to associate these attacks with prolonged use of the eyes at night and has therefore practically given up reading after dark. It is more than probable that the headache and the kidney disturbance are in both cases secondary to functional exhaustion of the posterior lobe of the hypophysis.

ILLUSTRATING DOCTOR WOODS'S ARTICLE ON "OCULAR ANAPHYLAXIS."

FIG. 1.

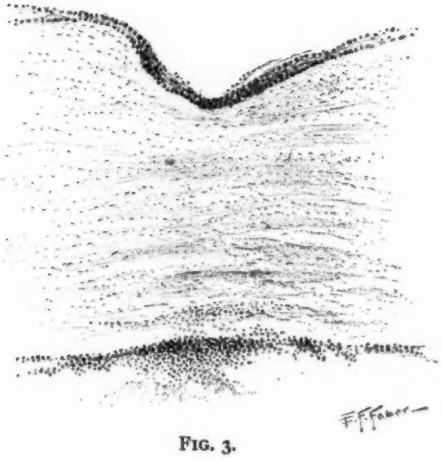

FIG. 3.

OCULAR ANAPHYLAXIS.

V. EXPERIMENTAL IRIDOCYCLITIS.

By Dr. ALAN C. WOODS, Philadelphia.

(*With three drawings on Text-Plates IX. and X.*)

(From the John Herr Musser Department of Research Medicine, University of Pennsylvania, Philadelphia.)

IN the previous papers (1) of this series, we have presented the evidence, theoretical and technical, which establishes a scientific foundation for the anaphylactic theory of sympathetic ophthalmia. In another paper (2) this evidence has been summed up, and one instance of apparent sympathetic ophthalmia in a dog, and presumably anaphylactic in character, is reported. It is the purpose of this paper to present further observations on the production of anaphylactic iridocyclitis, which apparently represents sympathetic ophthalmia in the dog.

TECHNIQUE.

The method employed has been to produce a hypersensitiveness of one eye by means of intraocular injection of homologous (dog's) uveal emulsion in the fellow eye. That such an ocular hypersensitiveness results from the injecting of uveal tissue in the fellow eye has already been demonstrated (1, 2). After a suitable interval, from two to three weeks, in which period sensitization of the second eye occurs, an "intoxicating" intraperitoneal injection of uveal emulsion has been given. Following Elschnig's observation (3) that sympathetic ophthalmia more frequently occurs in patients showing some other

underlying condition which lowers the natural resistance to disease, we have produced experimentally in several of these animals, morbid conditions of a non-infectious character, chiefly a uranium nephritis, or a phloridzin glycosuria.

Eleven dogs in all have been used in this section of our work. These dogs were carefully selected and were apparently free from all general disease. Before the primary intraocular injection, the eyes of all the animals were carefully examined to insure absence of ocular lesions.

CLINICAL MANIFESTATIONS.

After the primary intraocular injection, one of the dogs developed an infection in the injected eye. This dog was at once discarded, as introducing a complicating bacterial element. The remaining ten dogs were under observation for a period of over two months. The injected eyes of these dogs reacted with a more or less severe iridocyclitis which persisted during the period the dogs were under observation.

From within two to three weeks after the intraocular injection of uveal emulsion, the second, or uninjected, eye of these dogs began to show slight symptoms of irritation. These symptoms consisted in a moderate pericorneal injection, together with more or less pronounced photophobia. In those dogs which did not develop an outspoken iridocyclitis, these symptoms and signs of ciliary irritation in the second eye persisted for a period of a week or more, and then gradually cleared up; the eye becoming practically normal.

The "intoxicating" intraperitoneal injection was given from two to three weeks after the primary injection. In the dogs showing an anaphylactic iridocyclitis, an intensification of the symptoms in both the primary injected eye (an iridocyclitis) and in the second eye (symptoms of ciliary irritation) began to develop from within forty-eight hours to a week. In the injected eye the iridocyclitis present since injection became more severe. In the uninjected, or sympathizing eye, the symptoms of ciliary irritation became intensified, the pericorneal injection became more intense, the photophobia more marked, the pupil contracted practically to pin-point size, and then redilated slightly. The iris finally became immobile, slight exudates appeared in the anterior chamber, the edges

ILLUSTRATING DOCTOR WOODS'S ARTICLE ON "OCULAR ANAPHYLAXIS."

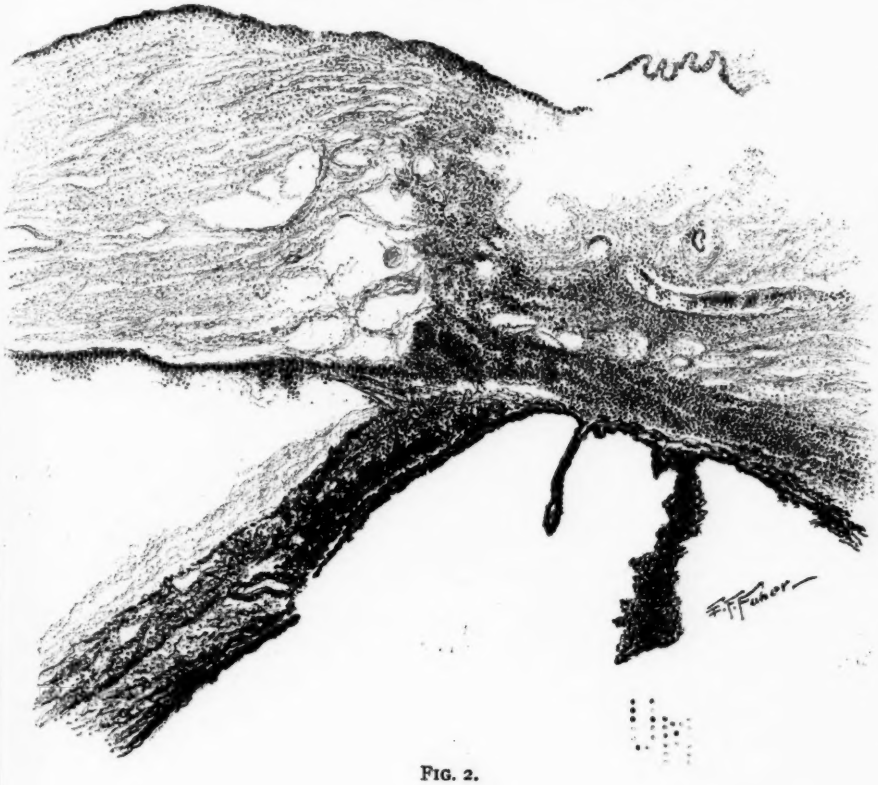

FIG. 2.

of the cornea became dull and grayish, vitreous opacities developed, and the tension of the globe became distinctly lowered. No definite fundus lesions were observed.

Of the ten dogs, one died from a uranium nephritis eight days after receiving the intoxicating dose. Three dogs developed outspoken disease in the second eye, which we believe to be sympathetic ophthalmia. The remaining six dogs developed symptoms of ciliary irritation in the second eye, which persisted for several weeks, with a gradual return to normal.

Of the three dogs developing the anaphylactic iridocyclitis, one has already been reported (2). This dog developed marked symptoms in the second eye within forty-eight hours after receiving the intoxicating dose. These symptoms consisted in pericorneal injection, photophobia, inactive pupil, exudation into the anterior chamber, vitreous opacities, and a diminution in intraocular tension. These symptoms grew rapidly more intense up to the death of the dog ten days later from an intercurrent lobar pneumonia. Histologically this eye showed a round-cell infiltration in the choroidal stroma, especially in the ciliary region, with a slight monocellular infiltration around the ciliary processes (Fig. 1).

The clinical histories of the remaining two dogs are as follows:

Dog 17–45. This is an apparently normal dog, whose eyes are entirely negative to external and ophthalmoscopic examination.

March 16. Under ether anæsthesia, the anterior chamber of the right eye was tapped and 1.0*cc* of dog's uveal emulsion was injected into the vitreous of this eye, by injection through the sclera, well back of the scleral-corneal margin.

March 24. The dog shows a very slight iridocyclitis of the right eye. The left eye is entirely clear.

March 30. The right eye shows a very slight iridocyclitis; the left eye is clear.

April 4. The condition of the right eye is unchanged; the left eye remains clear.

April 7. The right eye is in the same condition. The left eye to-day shows a slight pericorneal injection, the iris reacts and the media is clear. There is one small hemorrhage near the disk. On this date 9.7*cc* phloridzin solution[1]

[1] *1cc* phloridzin solution equals 0.2*gm* phloridzin in sodium carbonate solution. 1.0*cc* per kilo of body weight was given.

was given intravenously in attempt to establish an underlying disturbance, and 8.0*cc* dog's uveal emulsion given intraperitoneally to incite lesion in opposite eye.

April 9. The iridocyclitis present in the right eye is much more intense and the pupil is contracted to pin-point size. The left eye shows a small pupil and moderate pericorneal injection; the media is clear and the fundus negative.

April 12. The iridocyclitis is more marked in the right eye, as is also the pericorneal injection in the left eye; otherwise the condition is unchanged.

April 17. The process in the right eye is essentially as before. The left eye shows the pupil contracted to pin-point size and the pericorneal injection is quite intense. The small amount of the fundus that can be seen is negative.

April 20. The right eye is as described above. The left eye shows a violent pericorneal injection, the pupil is narrowed, the iris does not react to light, and the edges of the cornea are slightly hazy.

April 23. The right eye shows the same intense iridocyclitis, with a slight diminution on palpation in intraocular tension. The left eye shows a violent pericorneal injection and the tension is very low, the eye feeling quite soft. The lower part of the cornea is hazy, the iris is immobile and does not react. There is a small exudate over the dependent portions of the iris, vitreous opacities, and the fundus is not clearly seen. Photophobia is marked.

April 24. The right eye shows the same picture and is very soft. The left eye is in the same condition as yesterday, showing violent pericorneal injection, contracted and immobile iris, with a faint exudate in the anterior chamber, a hazy cornea, and vitreous opacities.

April 25. Both eyes are in the same condition, the intraocular tension is very low, both eyes being soft and mushy. The dog was killed with chloroform, autopsied, and the eyes and specimens of the tissues saved for section.

Dog 16–51. This is an apparently normal dog; both eyes are entirely negative to external and ophthalmoscopic examination.

March 16. Under ether anæsthesia the anterior chamber of the right eye was tapped, and 1*cc* of dog's uveal emulsion injected into the posterior chamber in the usual manner.

March 24. The right eye shows a slight anterior staphyloma, and a hemorrhagic area surrounding the cornea. There is present the usual evidence of an iridocyclitis. The left eye is clear.

March 30. The right eye is unchanged. There is the

faintest suggestion of pericorneal injection in the left eye. Otherwise it appears normal.

April 4. Both eyes are in the same condition as when last noted.

April 7. The right eye shows an iridocyclitis, with considerable lowering of the intraocular tension. The left eye to-day shows a moderate pericorneal injection. The iris appears normal, the media is clear, and the fundus negative.

8cc dog's uveal emulsion given intraperitoneally.

10.7cc phloridzin solution given intravenously.

April 9. The eyes appear in practically the same condition. The pericorneal injection in the left eye is possibly a little more intense.

April 12. The eyes continue in the same condition.

April 17. The right eye shows an intense iridocyclitis, moderate anterior staphyloma, and a diminution in intraocular tension. The left eye to-day shows a deep and intense pericorneal injection, the pupil is somewhat contracted, the media is clear, and the fundus appears normal. There is a definite photophobia.

April 20. The right eye is as described in last note but in the left the pericorneal injection and photophobia are both somewhat more marked.

April 23. The right eye is unchanged, but the left eye shows violent pericorneal injection, a contracted pupil, and the iris does not react to light. The edges of the cornea are slightly hazy, the media clear, and the fundus negative.

April 24. The right eye is unchanged. The pupil of the left eye is contracted to pin-point size and the condition described yesterday persists unabated.

April 25. The right eye shows a cloudy cornea with anterior staphyloma, an intense iridocyclitis, and a great diminution in intraocular tension. The left eye shows violent pericorneal injection, a hazy cornea, an inactive, lusterless iris, slight exudates in the anterior chamber, and vitreous opacities. The fundus is not clearly seen and the tension is distinctly lower. The photophobia is marked.

April 27. The condition of both eyes is essentially unchanged except that the intraocular tension appears lower. The dog was killed with chloroform, autopsied, and the eyes and specimens of the tissues preserved for section.

PATHOLOGY.

1. *General.*

17–45. At autopsy this dog showed a typical distemper broncho-pneumonia, characterized by the presence in the right lung of several discrete, elevated areas about 1.5*cm*

in diameter. Section through such areas showed a pneumonic process in the stage of red hepatization, extending out from the bronchus. The lower lobe of the left lung showed a larger pneumonic process involving half of the lobe. On section this appeared to be a fused broncho-pneumonia in the stage of red hepatization. All other tissues appeared normal.

The histological examination of the tissues of this dog showed a typical broncho-pneumonia in the right lung and the lower lobe of the left lung. In both kidneys there was a very slight pyelonephritis characterized by focal collections of mononuclear and polynuclear leucocytes along the papillary ducts in the pelvis. The other tissues all appeared normal.

16–51. This animal showed no evidence of any gross or histological change in any organ, except the eye.

2. *Special* (*Ocular*).

Gross. The gross appearance of the eyes of the two dogs was essentially the same. The right eye, which had been injected in each instance with uveal emulsion and which presumably acted as the exciting eye, had the following gross appearance. The cornea appeared normal. There was a faint fibrinous exudate in the anterior chamber and both the iris and the ciliary body appeared definitely swollen, the former being adherent to the anterior capsule of the lens. Over the ciliary body, extending forward in the posterior chamber and surrounding the lens, there was a web-like fibrinous exudate. The retina and choroid showed no gross lesions.

The gross appearance of the left, or sympathizing, eye was very similar in the two dogs. The cornea appeared to be slightly thickened. There was a slight exudate in the angles of the anterior chamber and in both dogs the iris was apparently thicker than normal. In dog 16–51, the iris was adherent to the anterior capsule of the lens by fibrinous synechiæ. In dog 17–45 synechiæ were not present. The ciliary body did not appear to be swollen but was covered, in both instances, by a fibrinous exudate, which extended forward in the anterior part of the posterior chamber, up to and surrounding the lens. There were no apparent gross retinal or choroidal lesions.

Microscopic. The pathological process in the eyes of both dogs was identical but appeared to be slightly more advanced in 17–45. The microscopic appearance of the eyes of this dog is described below.

Right (injected eye). The anterior segments of the

choroid, especially those parts underlying the ciliary processes, show a definite flat thickening. This thickening extends forward and involves the iris. Throughout the stroma of this section of the choroid, and the stroma of the iris, there is a moderate cellular infiltration, which appears to be mononuclear in type. In the anterior half of the posterior chamber there is a slight fibrinous exudate extending up to the margins of the lens. In this exudate are enmeshed a very few free pigment granules. The retina does not appear to be involved in the pathological process. The anterior chamber, Descemet's membrane, and cornea appear normal.

The left eye, which showed clinically the anaphylactic iridocyclitis, presented the following histological picture (Fig. 2). The anterior segments of the choroid, especially those portions underlying the ciliary processes, showed a marked thickening. The stroma of this section of the choroid is the seat of a dense mononuclear infiltration which extends forward and into the stroma of the iris, which is correspondingly thickened and shows the same cellular infiltration. The anterior endothelial layer of the iris is covered with a faint, homogeneous, eosin-staining exudate. The angles of the anterior chamber are involved in this same process and show a mononuclear exudate, which involves the ciliary body, and, extending forward, involves also the pectinate ligament, the lymph spaces, and Schlemm's canal, in which is a thin sero-fibrinous exudate. At this point, at the angle, there is a direct continuity between the infiltration of the choroid, the iris, and pectinate ligament. There is a moderate involvement of the ciliary processes, which are surrounded by a slight but definite mononuclear and fibrinous exudate.

The cornea is thickened, but the thickening appears due entirely to an interlamellar œdema, without the presence of any cellular elements. Descemet's membrane is, however, definitely involved in a process which is continuous with the involvement of the pectinate ligament. The endothelium shows a definite proliferation, and is covered with a faint, eosin-staining, homogeneous exudate, in which are enmeshed occasional lymphocytes (Fig. 3).

In the cellular infiltration of the stroma of the choroid, the mononuclear cells show occasional slight clumping, suggesting somewhat giant cells, but nowhere are true giant cells found.

The pathological process in the "sympathizing" eye is, in short, a chronic iridocyclitis, with involvement of the pectinate ligament, of the lymph spaces, and Schlemm's canal, and extending also to Descemet's membrane.

INFLUENCE OF OTHER MORBID CONDITIONS.

Elschnig (5) has commented upon the frequency, if not constancy, with which other pathological conditions, such as nephritis, diabetes, pulmonary diseases, etc., are encountered in patients with sympathetic ophthalmia. This observation has led him to suggest that such conditions may be predisposing factors in the development of sympathetic ophthalmia, after injury or disease of the exciting eye, insomuch as such diseases lower the general resistance and make the organism more subject to anaphylactic reactions.

Following this suggestion, several of the experimental dogs have been given subcutaneous injections of uranium nitrate, and intravenous injections of phloridzin, thus producing an acute nephritis or a phloridzin glycosuria. Two dogs with uranium nephritis died in ten days and one month respectively. The dog dying in ten days showed no symptoms of any kind in the second eye. The dog dying after a month showed a very slight sympathetic irritation which appeared about three weeks after the injection of uveal emulsion in the exciting eye and persisted without any increase in intensity up to the dog's death ten days later.

Three dogs were given injections of phloridzin. Two of these were the dogs showing the anaphylactic iridocyclitis.

Of more interest, however, were the pulmonary lesions in the dogs showing the experimental irido-cyclitis. One of the dogs showed a definite lobar pneumonia in the stage of gray hepatization. The cause of this pneumonia was not determined. Another dog showed what was apparently a typical distemper broncho-pneumonia. The lungs of the third dog were apparently normal. This pulmonary condition seems especially interesting, insomuch as it is well known that an anaphylactic reaction produces a definite pulmonary condition. It seems possible, although it is only a conjecture, that the resistance of the lungs to infection may have thus been lowered by the anaphylactic condition.

The second possibility is that the pneumonia was the primary process and that the ocular condition was secondary, due to bacterial metastasis. This possibility seems unlikely for the following reasons. The ocular condition in the three

dogs appeared clinically and histologically to be the same morbid process. Two of the dogs showed a pneumonia, the third dog appeared at autopsy to be entirely normal. The histological picture shown by the eyes of these dogs was in no sense the picture of a bacterial infection. The ocular process, although violent, leading to practical loss of the ball, involved only the uveal tract, and did not extend to any of the other tissues of the eye, which is not the course of a metastatic bacterial infection of the eye. Moreover, if the ocular condition were due to bacterial metastasis, there should be evidence of such metastasis in other organs. One dog, 17–45, showed, it is true, a slight pyelonephritis, but this in turn was probably secondary to the phloridzin injection. The remaining dogs showed no evidence of any bacterial metastasis. For these reasons, it appears probable to us that the ocular lesions were totally independent of the pulmonary lesions.

The three instances of anaphylactic iridocyclitis, apparently sympathetic in character, which are reported, occurred, therefore, either in dogs subjected to a phloridzin glycosuria, or in dogs which, during the period of observation, developed pneumonia. Five dogs, normal except for the sensitizing and intoxicating injections of uveal emulsion, developed no symptoms in the second eye, beyond the symptoms of a more or less mild sympathetic irritation. It seems probable, therefore, that Elschnig's suggestion that some other anomaly of the organism is needed to decrease the resistance, and so allow the development of sympathetic ophthalmia, is a correct view.

DISCUSSION.

The pathology of sympathetic ophthalmia has been the subject of much investigation, and it is generally conceded that the picture is fairly characteristic. According to Uhr (4), Ruge (5), and Fuchs (6) sympathetic ophthalmia is manifested pathologically as a chronic fibrino-plastic uveitis. The pathological changes are limited entirely to the uvea. Uhr describes the picture as characterized by a thickening of the choroid due to a monocellular infiltration. There is a predilection of this infiltration for the external layers of the

suprachoroid and occasionally a progression of the infiltration into the sclera, along the vessel walls, and along the lymph spaces of the ciliary nerves,—a process somewhat similar to a perivascular lymphangitis. Among the mononuclear cells are numerous mast cells, with epithelioid and giant cells, which may be massed in the infiltrated uvea as in a tubercle. Ruge emphasizes the fact that the disease is limited to the uvea, and is characterized by a monocellular infiltration, partially diffuse and partially circumscript, around the blood-vessels. Epithelioid and giant cells are present in the cellular infiltration and there is also a mononuclear infiltration around the ciliary process. This picture is essentially similar to that described by Fuchs.

In many respects the experimental iridocyclitis we have reported here resembles this histological picture. In other respects it is, however, different. The involvement of the pectinate ligament and Descemet's membrane is manifestly directly dependent upon the process in the iris and choroid, and is but a direct extension of this. The term "anterior uveitis" has indeed well been applied to this apparent corneal condition. In so far as this anaphylactic iridocyclitis is a chronic affair, characterized by a mononuclear infiltration, and limited to the uvea, it resembles the picture of human sympathetic ophthalmia described by Uhr and others. Moreover, the processes are similar in that both tend to involve the lymph spaces.

The processes differ in that the iridocyclitis in the dog involves especially, if not solely, the anterior portion of the uveal tract, while sympathetic ophthalmia in man involves especially the posterior portion of the uvea. However, in spite of this difference, the two conditions appear to us to be the same. The experimental condition appears to be sympathetic, following injury to the fellow eye. The same vital structure is involved in both conditions. The nature of the involvement is essentially the same in both conditions. Finally the difference in species may be sufficient to explain the difference in spite of the involvement in the uvea. To us the most important thing is that the same tissue in the dog and in man is involved in a similar pathological process.

Further minor differences are present, notably the occur-

rence of epithelioid and giant cells in the human disease, and the absence of these cells in the disease here reported. This last difference, however, we are inclined to minimize. It seems probable that the giant cells and epithelioid cells are in no way characteristic of the disease, but are merely an evidence of a greater chronicity, for, as a rule, the occurrence of such elements is but an evidence of chronicity—continued irritation. The resistance of dogs toward a chronic disease, the scarcity of such disease, and the difficulty, if not impossibility of producing chronic disease in dogs, is well known. Experimentally, as a rule, dogs respond to insults with an acute reaction, which speedily leads either to recovery, to loss of the organ involved, or to death, as the case may be. The most reasonable view, therefore, of the absence of epithelioid and giant cells in this condition in the dog and the occurrence of these elements in sympathetic ophthalmia in man, is that the anaphylactic iridocyclitis in the dog is more acute than the analogous process in man.

The early contraction of the pupil shown by these dogs is of especial interest. In the early studies in this series it was shown that when the eyes were perfused with specific antigen the pupil of a sensitized dog responded by a marked contraction. Further it is stated by Nettleship that oscillation of the pupil is one of the earliest manifestations of impending sympathetic ophthalmia.

In reporting this experimental condition it is realized that it is not a typical sympathetic ophthalmia, as the term is used clinically. Working with lower animals, it is almost hopeless to expect a typical reproduction of such a disease. Yet that this condition is sympathetic ophthalmia in the dog, we do believe. It follows injury to the fellow eye where uveal tissue is liberated for absorption, and is produced by an anaphylactic mechanism. The process in the sympathizing eye is limited to the uvea. The underlying pathological nature is similar to that of sympathetic ophthalmia as we recognize it in man.

If our supposition is true, that this anaphylactic iridocyclitis is a true sympathetic ophthalmia in the dog, it is the last step in the experimental demonstration of the anaphylactic nature of sympathetic ophthalmia. We believe the questions of ocular anaphylaxis, the antigenic properties of uveal pigment,

and the experimental reproduction of a sympathetic ocular disease in dogs by anaphylactic means, to be established. Whether or not these purely laboratory and experimental findings can be brought in relationship with clinical sympathetic ophthalmia remains to be seen. We fully appreciate that the final demonstration of the true nature of sympathetic ophthalmia, or indeed of any disease, must be in man.

SUMMARY.

An anaphylactic iridocyclitis has been produced in dogs by sensitization through intraocular injections of dog's uveal emulsion in the fellow eye, and intoxication through intraperitoneal injection. The condition begins with signs of ciliary irritation, followed, in such dogs as develop the disease, by an outspoken iridocyclitis, with subsequent descemetitis. Three out of ten animals developed such a condition. Histologically the process is a thickening and round-cell infiltration of the anterior portion of the choroid, and the iris, with an involvement along the pectinate ligament, of Schlemm's canal, and a descemetitis. From the mode of occurrence, character, and pathology of the disorder, it is believed to be sympathetic ophthalmia in the dog.

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THE SURGICAL TREATMENT OF CORNEAL SUPPURATION IN EXOPHTHALMIC GOITER.[1]

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SLIGHT corneal involvement in exophthalmic goiter is easily controlled by care and simple remedies. The severer corneal lesions, particularly when they occur in the toxic cases, are desperate and tax the surgeon's resources to the utmost. The loss of both corneæ in exophthalmic goiter is not so very unusual; Sattler, in 1907, collected the reports of forty cases where both eyes were lost. The following case of exophthalmic goiter where one eye was lost and the other eye was saved with some vision may be of interest.

B. K., aged 35, stated that his eyes began to become prominent in August, 1916. His general condition during the past six months has been very much affected, and he has lost forty pounds in weight. On Jan. 13, 1917, the patient came to the Herman Knapp Memorial Eye Hospital on account of pain in the left eye. Both eyes were very prominent; the right eye could be closed only with effort, and at night this eye was kept open; there was some conjunctival congestion. The conjunctiva of the left eye was red and chemosed, the cornea was infiltrated with pus and was dried out, a little clear cornea remained in the upper part. Vision: R = 20/30; L = 1/200.

The patient was restless, anxious, with occasional tremor and a dry skin. Pulse about 90. The thyroid gland was hard, fibrous, but not enlarged. No difficulty on swallowing. He had a heart lesion and weighed 115 pounds. Dr. John Rogers of New York was kind enough to see the patient and prescribed adrenalin and advised the ligation of the

[1] Read at meeting of American Academy of Ophthalmology and Oto-Laryngology at Pittsburg, October 29–30, 1917.

left inferior thyroid artery. This operation was done. During the next few days his general condition remained about the same; at times he was alert and quiet, at other times he was restless, apathetic and could not sleep at night, and had no appetite. He had no diarrhœa. The congestion of the right eye was relieved and the cornea remained intact. The process in the left eye continued and the cornea sloughed. Jan. 20th: blood-pressure 104–110; weight 110½ lbs. As the patient was losing weight steadily, Dr. Rogers advised transferring him to the St. Francis Hospital, where he was better prepared to ligate the remaining thyroid arteries, which was then done.

On Feb. 5th the right eye had again become inflamed and a part of the outer wall of the orbit was resected by Dr. Rogers to relieve the intraorbital pressure. I saw the patient on Feb. 11th, as the right eye was not doing well. I found the right conjunctiva chemosed and swollen and the cornea in its lower half infiltrated with pus. The patient was immediately returned to the eye hospital and an attempt was made to arrest the process in the right cornea by frequent irrigation, applications of argyrol salve, and bandaging. On Feb. 12th the corneal condition was worse: the part of the cornea above, which had remained clear, had now become clouded. There was very little discharge. The general condition of the patient was about the same, except that he had lost ten additional pounds.

Under ether it was decided to suture the eyelids, following out the suggestion of Priestly Smith mentioned in the discussion of Juler's article[1] in the *T. O. S.*, 1913. An intermarginal incision was made in both lids, splitting the lids rather deeply. Mattress sutures were then introduced from above through the cut part of the lids, drawing together the bottoms of the two grooves, and the lids were approximated without much tension throughout a broad contact of the cut surfaces. Cutaneous incisions were made along the margins of the orbit above and below and on the temporal side in order to relieve the tension of the skin, as suggested by Bishop Harmon.[2] The left eye was eviscerated. At the following daily dressings the lids of the red eye looked normal, though somewhat bulging, and the sutures were apparently holding. The conjunctival sac was irrigated daily from the sides. The patient's general condition was about the same. A small opening formed at the center of the lids through which the progress of the corneal lesion could be watched. The upper part of the cornea cleared, a

[1] Juler, F. A., "Acute Purulent Keratitis in Exophthalmic Goiter," *T. O. S.*, 1913, p. 71.

[2] *L. c.*, p. 74.

small perforation took place below, and a necrotic plug was cast off. On Feb. 20th his weight was 91½ lbs. On the fifth day the sutures were removed on account of stitch-infection, the lids however remained united. The patient was put on thyroid residue in addition to the suprarenal extract, and his general condition began to improve. The upper third of the cornea remained clear. Several decayed teeth were extracted, and the general improvement continued. The points of union of the lids slowly stretched, forming bands 2–3*mm* long, the cornea nevertheless was sufficiently protected. The general health showed steady improvement, but there was no recession of the exophthalmus. The bands uniting the lids were divided and this fall an optical iridectomy was performed upward; the outer third of the palpebral fissure was sutured to facilitate closing the lids. The vision was 6/200.

This desperate case is instructive from a number of points of view. It shows first that there is no general treatment which will lessen the exophthalmus promptly enough to safeguard the affedcte cornea. C. H. Mayo and others recommend the resection of the superior sympathetic ganglion under these conditions. This procedure probably offers the most hope though the operation is not simple, and our patient's poor general condition did not warrant so severe a surgical intervention. Juler (*l. c.*) has investigated the results of sympathectomy, and states that Balacescu[1] found that after operation out of 41 cases the exophthalmus was improved in 34 after a few hours or days, in 3 cases one eye only was improved, in 4 there was no effect. Later results were given in 25 cases, and marked improvement and even disappearance of the symptoms were observed in 19, no change in 3, increase in 3. In Juler's own case no marked recession of the exophthalmus occurred after this operation. In other words, favorable influence on the exophthalmus is by no means obtained in all cases. At the same time, in view of the desperate nature of the condition, if the patient's condition permits, this operation should be attempted.

The local condition remains the important one to correct, and if this can be relieved the corneal process will be arrested. The important contributing cause is the chemosed and thick-

[1] Balacescu, *Arch. f. klin. Chirurgie*, 1912.

ened conjunctiva in the lower half of the eyeball, which mechanically prevents the closure of the lids. The lids must be brought together to cover the cornea. Suture of the lids has undoubtedly been practiced by everyone for this purpose, but ordinary suturing even with freshening of the lid-margins is not satisfactory, as the sutures do not hold. In my opinion, the suture method of Priestly Smith, augmented by the releasing cutaneous incisions of Bishop Harmon, meets these conditions best, and the favorable result obtained in this case must be ascribed to it.

NARROWING OF THE PUPIL DOES NOT LOWER NORMAL INTRAOCULAR TENSION (100 CASES).[1]

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THESE observations were made to determine whether or not there is a lowering of normal intraocular tension when the pupils are made smaller. The necessity for determining this matter arose from our routine clinical work on three types of cases.

1st. "Borderline Glaucoma" cases.

In these cases we were dealing with slightly enlarged pupils, suspicious cups, inconclusive narrowing of fields, and a tension above, say, 25*mm* Hg. We often noted a definite lowering of tension after a miotic, while at other times there was no change.

2d. Definite "high tension" simple glaucoma in one, with tension well within so-called normal limits in the other eye. The tension frequently fell in this second eye though in some cases it remained unchanged after miosis (Storey).

3d. Acute inflammatory glaucoma in one eye with normal tension in the other eye unaffected by narrowing of the pupil (Field).

It is at once evident that in these types of cases it is of prime importance to know whether or not normal tension is reducible by miotics.

The material consists of 100 cases studied at the Cook

[1] From the Research Laboratories of Cook County Hospital and Eye Clinic of Dr. E. V. L. Brown.

Read before the Chicago Ophthalmological Society, November, 1917.

County Hospital. They were patients that gave no history of eye trouble and were not seriously sick. Their ages ranged from 12 to 67 years. Only cases in which satisfactory tonometer readings could be made were used.

As in a previous series of cases,[1] all tension readings were taken with the Schiotz tonometer, and holocain in 1% sol. was used as an anæsthetic. The size of the pupil was first noted—then the tension taken. One half hour later the tension was again taken. The pupils were then rather quickly contracted with eserine salicylate in 1 or 2% sol. The average size of the pupils after the miosis was 1¼*mm*. All pupils were contracted to at least 2*mm*. Holocain was again instilled and the final tonometric readings taken.

In no case was there a lowering of tension following the contraction of the pupil greater than 3mm Hg.

This amount is well within the recognized limits of error of observation in the use of the tonometer. There was in no case any difference between the first and second readings before the miosis.

Following the use of the eserine many patients of course complained of diminution of vision and headache; a few of pain within the eyes; a few became nauseated; while four or five vomited. The pupils remained contracted a variable length of time—in a few eyes as long as seventy-two hours.

From this series of 100 cases we may therefore conclude that: intraocular tension in normal eyes is not to be lowered by the narrowing of the pupil. The corollary of this statement, namely, that a decrease of intraocular pressure following the use of a miotic necessarily means that an eye is glaucomatous, is not even herewith postulated and does not properly come within the scope of this paper. We can merely verify the statement frequently made—Narrowing the pupil does not lower normal intraocular tension.

[1] "The Effect of Tonsillectomy on Normal Intraocular Tension" (100 cases), ARCH. OF OPHTH., vol. xlvii., No. 1, p. 46.

TABLE.

Case No.	Age.	Before Miosis.						After Miosis.			
		Pupils.		Tension.				Pupils.		Tension.	
				First.		*½ hour later.*					
		R. *mm*	L. *mm*	R. *mm*	L. *mm*	R. *mm*	L. *mm*	R. *mm*	L. *mm*	R. *mm*	L. *mm*
1	27	4	4	14¼	13¼	14¼	12¼	1	1	15¼	13¼
2	44	2½	2½	12¼	16½	14¼	16½	1	1	13¼	15¼
3	39	2½	2½	16½	16½	16½	16½	1	1	18	18
4	25	4	4	13¼	13¼	13¼	14¼	1¼	1¼	13¼	13¼
5	21	4	4	18	19½	18	19¼	1½	1½	19½	18
6	23	5	5	18	18	16½	15¼	1¼	1¼	18	18
7	30	6	5	12¼	11¼	12¼	13¼	1	1	14¼	14¼
8	31	3½	3½	19½	19½	21½	18	1¼	1¼	19½	18
9	36	3	3	16½	16½	16½	15¼	1	1	18	16½
10	57	5	5	16½	16½	15¼	16½	1¼	1¼	15¼	18
11	56	3½	3½	13¼	13¼	12¼	13¼	1½	1½	13¼	13¼
12	72	4	4	18	18	18	21	2	2	18	18
13	39	4	4	15¼	16½	15¼	15¼	1	1	15¼	16½
14	37	4	4	13¼	14¼	13¼	14¼	1½	1½	13¼	15¼
15	41	4	4	18	19½	18	19½	1½	1½	19½	19½
16	65	5	5	21	23	23	23	1½	1½	21	21
17	61	4	5	14¼	14¼	14¼	14¼	1	1	14¼	13¼
18	35	5	5	14¼	14¼	13¼	14¼	1½	1½	14¼	14¼
19	49	5	5	14¼	14¼	14¼	14¼	1	1¼	14¼	15¼
20	25	4	4	12¼	12¼	12¼	13¼	1	1	12¼	14¼
21	21	4½	4½	13¼	14¼	13¼	14¼	1	1	11½	11½
22	24	4	4	14¼	16½	15¼	15¼	1	1	13¼	13¼
23	15	3½	3½	13¼	13¼	13¼	13¼	1	1	13¼	12¼
24	14	3	3	19½	19½	19½	19½	2	1½	19½	18
25	43	3	3	16½	16½	16½	15¼	1	1¼	14¼	15¼
26	38	4	4	13¼	13¼	14¼	12¼	1½	1½	14¼	15¼
27	28	4	4	12¼	13¼	13¼	12¼	¾	¾	13¼	13¼
28	21	4½	4½	18	18	18	16½	1¼	1¼	19½	18
29	53	3	3	18	16½	16½	16½	1	2	18	19½
30	23	3½	3½	15¼	16½	18	19½	1¼	1	18	16½
31	55	4½	4½	16½	16½	14¼	16½	1½	1½	13¼	14¼
32	19	3½	3½	16½	15¼	18	15¼	1½	1½	18	18
33	51	4	3	15¼	16½	15¼	16½	1	1	14¼	15¼
34	76	4½	4½	21	21	21	19½	1½	1½	21	18
35	49	4	3½	16½	16½	16½	14¼	1½	1	16½	13¼
36	12	4	4	15¼	14¼	13¼	16½	1½	1½	14¼	15¼
37	62	4½	4½	14¼	14¼	16½	15¼	1¾	1¼	14¼	13¼
38	12	4	4	10	10	10	10	2	2	12¼	11¼
39	30	4½	5	13¼	12¼	14¼	12¼	1¼	1¼	13¼	10
40	18	4	4	18	19½	18	18	1	1	16½	16½
41	46	3	3	16½	16½	18	16½	1	1	16½	16½
42	48	4	4	18	16½	18	18	1	1	18	18
43	64	4	4	14¼	15¼	15¼	15¼	1	1	14¼	14¼
44	39	3	3	15¼	15¼	15¼	15¼	1½	1½	14¼	15½
45	35	5	5	18	18	18	18	1½	1½	18	18

Case No.	Age.	Before Miosis.						After Miosis.			
		Pupils.		Tension.				Pupils.		Tension.	
				First.		*½ hour later.*					
		R. *mm*	L. *mm*	R. *mm*	L. *mm*	R. *mm*	L. *mm*	R. *mm*	L. *mm*	R. *mm*	L. *mm*
46	68	4	4	15¼	14¼	16½	16½	1	1	14¼	16½
47	36	5	5	18	18	16½	18	1	1	19½	18
48	32	4½	4½	15¼	16½	16½	16½	2	2	18	16½
49	36	4	4	13¼	13¼	12¼	14¼	1½	1½	12¼	13¼
50	33	4	4	15¼	15¼	15¼	15¼	2	2	15¼	14¼
51	33	4¾	4¾	10¾	10¾	12¼	11¼	1¼	1½	11¼	12¼
52	74	4	4	18	18	18	16½	1	1	16½	18
53	77	5	5	18	18	18	18	1½	1½	16½	16½
54	51	5½	5½	21	21	23	21	1	1	23	23
55	17	4	4	19½	18	19½	18	1	1	18	18
56	35	4½	4½	16½	16½	16½	18	1¾	1	15¼	16½
57	35	4	4	16½	14¼	15¼	15¼	2	2	13¼	12¼
58	27	4½	4½	14¼	14¼	13¼	14¼	1½	1½	12¼	13¼
59	32	3	3	13¼	12¼	14¼	14¼	1½	1½	12¼	11¼
60	21	3½	3½	19½	19½	19½	19½	1¼	2¼	19½	21
61	40	4	4½	14¼	14¼	13¼	13¼	1	1¼	14¼	11¼
62	25	3	3	16½	16½	16½	15¼	1	1	18	16½
63	22	4	4	18	18	18	18	¾	¾	18	16½
64	29	5	5	16½	16½	15¼	16½	1¼	1¼	15¼	18
65	22	3½	3½	13¼	13¼	11¼	13¼	1½	1½	13¼	13¼
66	14	4	4	18	18	18	21	2	2	18	18
67	28	4	4	15¼	16½	14¼	15¼	1	1	15¼	16½
68	40	4	4	13¼	14¼	14¼	14½	1¼	1¼	14¼	15¼
69	41	3	3	19½	19½	18	19½	1½	1½	19½	21
70	29	4	5	14¼	14¼	14¼	14¼	1	1	14¼	13¼
71	22	5	5	23	25	21	25	1½	1½	21	25
72	21	5	5	14¼	14½	14¼	14¼	1½	1½	14¼	13¼
73	42	4½	5	14¼	13¼	14¼	14¼	1	1¼	14¼	13¼
74	37	5	5	9¼	9¼	10	9¼	1	1	9¼	10
75	39	4	4	12¼	12¼	12¼	13¼	1	1	14¼	14¼
76	19	6½	6½	15¼	15¼	15¼	15¼	1¾	1¾	15¼	15¼
77	37	4	3	11½	11½	12¼	12¼	1	1	13¼	12¼
78	57	4½	4½	15¼	14¼	15¼	15¼	1½	1½	15¼	14¼
79	25	3	3	15¼	18	18	18	1¼	1¼	16½	18
80	35	3½	3½	13¼	13¼	14¼	14¼	1¼	1¼	15¼	14¼
81	36	5½	5½	15¼	15¼	14¼	15¼	1	1	15¼	15¼
82	32	4½	4½	14¼	14¼	14¼	14¼	1½	1½	14¼	14¼
83	40	5	4½	12¼	13¼	12¼	12¼	1¼	1¼	11¼	12¼
84	41	5	5	16½	16½	15¼	14¼	1¼	1¼	15¼	15¼
85	22	5	5	8½	9½	8½	9½	1	1	8½	10
86	28	3½	3½	18	18	18	18	1½	1½	18	18
87	47	4½	4½	19½	19½	18	19½	1	1	19½	19½
88	19	3	3	14¼	15¼	15¼	14¼	1½	1½	14¼	14¼
89	27	4	4	16½	16½	16½	16½	1¼	1¼	16½	16½
90	23	4	4	14¼	15¼	15¼	15¼	2	2	14¼	15¼
91	39	4	4	18	18	18	18	1½	1½	18	18
92	55	3	3	19½	21	19½	21	¾	¾	18	21

Case No.	Age.	Before Miosis.						After Miosis.			
		Pupils.		Tension.				Pupils.		Tension.	
				First.		*½ hour later.*					
		R. *mm*	L. *mm*	R. *mm*	L. *mm*	R. *mm*	L. *mm*	R. *mm*	L. *mm*	R. *mm*	L. *mm*
93	47	5	5	18	18	16½	16½	1	1	16½	16½
94	50	4½	4½	15¾	15¼	15¼	15¼	1	1	14¼	13¾
95	33	3	3	18	18	19½	18	¾	¾	16½	16½
96	28	4	4	14¼	13¾	14¾	14¼	1	1	13¼	12¼
97	31	4½	4½	16½	16½	16½	16½	1	1	16½	16½
98	61	5	5	16½	14¾	16½	16¼	1¼	1¼	14¼	13¼
99	41	4½	4½	16½	16½	18	18	1½	1½	16½	16½
100	45	3½	3½	15¼	14¼	16½	16½	1½	1½	15¼	16½

DENTAL INFECTION IN EYE DISEASES..

By Dr. WILLIAM F. C. STEINBUGLER, New York.

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WITHIN the last few years much has been written concerning the relation between dental infection and the condition generally described as "rheumatism." It has been definitely proven that various forms of arthritis are cured or materially benefited by the extraction of infected teeth or by the cleaning up of a pyorrheal condition of the mouth. The arthritis occurs, in all probability, as the result of the lodging in the joint of bacteria or of their products which have entered the blood stream from the mouth. This would explain the migratory phase of "rheumatism," distant joints being affected in rather rapid succession, and would disprove the old theory of rheumatism being due to a non-assimilation of the acid products of proteid metabolism.

As would naturally be supposed, these organisms also lodge in the eye, giving rise to infection there. In fact this is the case more frequently than has heretofore been believed. It has been estimated that 50% of all adults suffer from pyorrhea in some form and, in reporting a series of 215 eye infections due to sepsis, Lang (1) found that 139, or nearly 65%, were due to this cause. Any part of the eye may be attacked as the result of dental infection, although by far the greatest number of cases show the iris, ciliary body, or choroid to be affected. Another condition of the teeth which may give rise to infection of the eye is an apical or so-called "blind" abscess. These abscesses are quite common, Ulrich (2) having found that apical abscesses were present in 83% of dead teeth and in over 68% of artificially devitalized teeth, and their

presence is often unsuspected or undetected until the X-rays are employed. Brown and Irons (3) in their excellent paper on "The Etiology of Iritis," found dental infection to be the source of iritis in 18 of the 100 cases studied, and 41 of the cases showed apical infection or alveolar abscesses on X-ray examination, which findings were subsequently confirmed by the oral surgeon. In their study peridental infection was not included. This would exclude pyorrhea alveolaris, which according to Finnoff (4) is the most frequent cause of ocular lesions, ranking higher than blind abscesses, septic roots, or periapical abscesses. Opposed to this view is Lawler (5) who maintains that pyorrhea itself is a very infrequent cause of ocular infection. He bases his opinion on the facts that careful treatment of the mouth has failed to exert any favorable action upon the ocular condition, especially in cases of scleritis and sclero-keratitis, and that vaccine treatment has been generally disappointing.

The exciting organism of ocular infections due to oral sepsis may be any one of the variety or a combination of those found in the mouth or on the tooth roots. It is obviously impossible in most cases to determine the exact organism causing the infection in the eye as an eye is seldom enucleated as the result of such an infection, but from cultures obtained from the roots of teeth, it is fair to assume that streptococci, especially the streptococcus viridans, staphylococci, and pneumococci are the bacteria most frequently involved. Hartzell (6) has shown that the streptococcus viridans is present in chronic dental abscesses in pure culture and also in pyorrhea. Hæmolytic streptococcus is absent. These organisms have never been found in healthy tissue. In studying a series of forty dental abscesses, Gilmore and Moody (7) found that streptococci were the organisms most constantly present and Hartzell and Henrici (8) maintain that streptococci of the viridans group are always present in peridental suppurative lesions. They are normally of low virulence but are at times able to produce lesions of the heart, aorta, kidneys, and joints. They are constantly present in lesions presenting a large ulcerated surface through which they may pass into the deeper tissues and the blood stream and cause metastases. The exact identity of the streptococcus viridans is still in

doubt. Hartzell of the University of Minnesota considers the streptococcus viridans of Schottmueller to be the same organism as the streptococcus brevis of Van Lingelsheim, the streptococcus salivarius of Andrewes and Horden, and to be identical with the diplococcus rheumaticus of Poynton and Paine. Edward C. Rosenow of the Mayo Foundation is a radical opponent of this theory. He asserts that the streptococcus viridans can change into the streptococcus pyogenes on the one hand and the pneumococcus on the other. The pus-producing cocci are not permanent inhabitants of the mouth. Their presence is considered accidental. Moody found the staphylococcus aureus eighty times in one thousand mouths, but found the staphylococcus pyogenes albus which possesses low virulency frequently in suppurating conditions of the mouth. The so-called "blind" abscesses at the apex of a tooth may become purulent by the staphylococci gaining entrance to that region. Henrici has attempted to demonstrate the pathogenicity of dental streptococci by complement fixation test, as has been done by Besredka, Hastings, and others. He concludes that the results have been negative because the patients do not produce antibodies in sufficient quantity to be demonstrated by the method of complement fixation and that unless the local foci are removed a vaccine will hardly be of benefit.

A most interesting phase of infection is the selective action possessed by bacteria and this has been very carefully studied by Rosenow. He found that streptococci obtained from the appendix if injected into animals most often produced lesions in that organ. Also, that streptococci isolated from arthritis more often affected the eye than those isolated in gastric and duodenal ulcers and that organisms from the joint show a selective action for the eye. The selective tissues of mouth organisms have been the joints, endocardium, and eye. Irons (9) in studying a series of 329 unselected cases variously grouped as to disease, X-rayed 124. In 44% of these 124 alveolar abscesses were found. In the arthritic group alveolar abscesses were present in 76%; in the nephritis-cardio-vascular group they were present in 47%; and in the group comprising other diseases, such as pneumonia, respiratory and gastro-intestinal, 23%. Abnormalities in tonsils were present

in 45% of the arthritic group, in 24% of the nephritis-cardio-vascular group, and in 19% of the remainder. Other chronic conditions such as sinus infections or infections of the genito-urinary tract were present in 21% of the arthritic group, 13% of the nephritis-cardio-vascular group, and in 11% of the other diseases. Syphilis was found in 23% of the arthritic group, 39% of the nephritis-cardio-vascular group, and 13% of other diseases. He considers the use of vaccines doubtful, at best being only an adjunct to other forms of treatment. From these figures it would seem that while there may not be a direct relationship between alveolar abscesses and the various diseases enumerated, the percentage of coincidence is far greater.

The exact mode of eye infection from a dental focus is still a debated question. Numerous theories have been advanced. Lelongt holds that infection takes place through channels in the bone, subperiosteal tissue, or lymphatics. Polet thinks that in addition to extension through the subperiosteal and osseous channels infection is transmitted along the nerves. Dutoit believes that infection can be carried from the dental alveolus through the alveolar plexus and the pterygoid plexus into the ophthalmic vein and cavernous sinus. Ulrich (2) and Dimmer (10) believe that the infection is always hematogenous and Finnoff regards this as the only possible mode of deep-seated infection as the lymphatics of the mouth drain into the mandibular and cervical glands. A direct extension through the venous channels from the diseased area to the cavernous sinus is anatomically possible and unquestionably has produced a sinus thrombosis and its symptoms.

There is no definite clinical picture that can be attributed to dental infection. Any part of the eye may be affected, but the iris, ciliary body, and choroid are most frequently involved. Ibershoff (11) states that "the cases can be classed as subacute and chronic serous cyclitis with the formation of exudates on the posterior layer of the cornea, in the aqueous and vitreous. The cases are long-standing, mild in character, devoid of pain or other manifestations associated with acute inflammatory processes, and have involved the iris but little or not at all." This represents a true picture of dental infection with the possible exception of frequency of iris involvement. The iris is probably more frequently affected than

would be supposed from his statement. While a purulent exudate is at times present, it is exceptional. In a diffuse suppurative pyorrhea with an abundance of pus in the mouth it is only logical to assume that the accompanying eye infection would also be of a purulent type. The reason why this is rarely so is explained by de Schweinitz (12) who states that "although the primary lesion is suppurative it produces a non-septic focus because the organisms decrease in virulence in their contact with the blood stream and they are not contained in emboli or blood clot."

Since the establishment of a dental department at the Herman Knapp Memorial Eye Hospital two years ago the investigation of dental infection has received particular attention and the following report represents the work of the first year and a half. The work was carried on in collaboration with Dr. Joseph M. Levy, who performed the dental work, and Dr. Marshall Carleton Pease, Jr., who performed the bacteriological work. In all fifty-three cases were examined, the diagnosis of these cases being as follows: acute iritis and iridocyclitis, 13; acute and chronic choroiditis, 10; chronic iridocyclitis, 9; post-operative iritis, 4; detachment of retina, 4; episcleritis, 2; dendritic keratitis, 2; and one case each of chorioretinitis, chronic cyclitis, irido-choroiditis, interstitial keratitis (non-specific), vesicular keratitis, keratoiritis, retrobulbar neuritis, retinal hemorrhage, and ulcer of the cornea. The routine treatment of cases where other infections are excluded is to have the teeth X-rayed, the blood examined, the streptococcus complement fixation test being employed, the teeth treated and, if necessary, extracted, and a vaccine prepared from the culture obtained at the time of extraction.

In examining this series of cases fifty-two of the fifty-three patients showed dental infection on the same side as the affected eye. This would lead to the belief that infection does not occur through the blood stream, as if this were so it would seem that more cases in which dental infection was present on the opposite side of the affected eye would have been noted. In view of this observation it is a logical conclusion that infection is carried either by means of the lymphatics or through osseous channels.

It was found that of the 53 cases 15 were mixed infections. One patient, having a + + + + Wassermann reaction in addition to infected teeth, was treated with mercury with no improvement. As soon as dental treatment was instituted the condition cleared up rapidly. Two cases showed a positive Wassermann reaction and a reaction to tuberculin besides having infected teeth. In the one having a + + + + Wassermann no improvement took place as the result of specific or dental treatment. In the other having a + + Wassermann reaction no mercury was given, but improvement took place as the result of dental treatment. The remaining 12 cases reacted positively to tuberculin in addition to having infected teeth. Of these, 6 received both tuberculin injections and dental treatment, 3 cases showing no improvement at all, 2 showing improvement as the result of dental treatment, and in 1 case it was impossible to say whether the dental treatment or the tuberculin injections caused the improvement. Of the remaining 6 cases 2 were improved as the result of dental treatment and 4 showed no improvement whatsoever.

The streptococcus complement fixation test was employed in 40 cases, being positive in 24 or 60% and negative in 16 or 40%. Of the 24 giving a positive reaction, 4 or 16 + % were cured; 14 or 58 + % were improved, and in 6 or 25% there was no improvement. Of the 16 cases giving a negative reaction, 3 or 18 + % were cured; 8 or 50% were improved, and in 5 or 31 + % there was no improvement. Of the 4 cured cases giving a positive reaction 2 showed streptococcus viridans in the cultures and received vaccines, no cultures being made in the remaining 2. Of the 14 improved cases giving a positive reaction 4 showed streptococcus viridans in culture, 1 of whom received vaccines; 2 showed staphylococcus in culture and received vaccines; 1, a case of severe pyorrhea, received stock vaccines, and in the remaining 7 cases no cultures were made. Of the 6 cases giving a positive reaction and showing no improvement, 2 showed streptococcus viridans in culture and received vaccines, no cultures being made in the remaining 4 cases. Of the 3 cured cases giving a negative reaction, 1 showed streptococcus viridans in culture but received no vaccines; in the other 2 cases no cultures were

made. Of the 8 cases showing improvement and giving a negative reaction, 4 showed streptococcus viridans in culture, 2 of whom received vaccines, and in the remaining 4 cases no cultures were made. Of the 5 cases giving a negative reaction and showing no improvement, 3 showed streptococcus viridans in culture and received vaccines, and in the remaining 2 no cultures were made.

In 25 cases bacteriological cultures were made either from the roots of the teeth extracted or from the tooth sockets. 14 or 56% showed a pure culture of streptococcus viridans; 2 or 8% showed a pure culture of staphylococcus; 4 or 16% showed a mixture of streptococcus and staphylococcus; 3 or 12% showed a mixture of streptococcus, staphylococcus, and bacillus pyocyaneus; 1 or 4% showed a mixture of streptococcus, staphylococcus, and pneumococcus, and 1 or 4% showed a mixture of streptococcus, staphylococcus, bacillus pyocyaneus, and pneumococcus. In analyzing the 14 cases in which a pure culture of streptococcus viridans was obtained it was found that 10 were of a chronic nature, the remaining 4 being made up of 3 cases of post-operative iritis and 1 case of acute iritis. This would seem to bear out Hartzell's theory that the streptococcus viridans is an organism of diminished virulence. Of the 2 cases which showed a pure culture of staphylococcus, 1 was an acute iridocyclitis, the other an exudative choroiditis. Of the 4 cases showing both streptococcus and staphylococcus, there was one each of detachment of the retina, acute iritis, post-operative iritis, and retinal hemorrhage. Of the 3 cases showing streptococcus viridans, staphylococcus and bacillus pyocyaneus, there were 2 cases of exudative choroiditis and one of chronic iridocyclitis. The case showing streptococcus, staphylococcus, and pneumococcus was one of episcleritis, and the case showing streptococcus, staphylococcus, bacillus pyocyaneus and pneumococcus was one of exudative choroiditis.

Vaccines were administered to 18 patients, 17 receiving autogenous vaccines and one stock vaccines. 2 or 11 + % of these cases were cured; 10 or 55 + % were improved, and in 6 or 33 + % there was no improvement. Both the cured cases gave a positive streptococcus complement fixation test, 1, a case of chronic iridocyclitis, showing a pure culture of

streptococcus viridans: the other, a case of episcleritis, showing streptococcus, staphylococcus: and pneumococcus in the culture. Of the 10 cases showing improvement, 2 were cases of post-operative iritis, showing a positive complement fixation test and a pure culture of streptococcus viridans; 2, cases of chronic iridocyclitis, 1, in which the complement fixation test was not made, showing streptococcus, staphylococcus, and bacillus pyocyaneus in culture, the other, in which the complement fixation test was negative, showing streptococcus viridans in culture. The other cases were: acute recurrent iritis, in which the complement fixation test was not made, but which showed streptococcus viridans and staphylococcus in culture; vesicular keratitis, with a negative complement fixation test, and showing streptococcus viridans in culture; a case of acute iridocyclitis with a positive complement fixation test, showing staphylococcus in culture; a case of corneal ulcer with a positive complement fixation test and which received stock vaccines (staphylo-acne); a case of chorioretinitis, in which the complement fixation test was not made, showing streptococcus viridans in culture, and a case of choroiditis with a positive complement fixation test, showing staphylococcus in culture. Of the 6 cases in which no improvement was obtained as the result of vaccine treatment, there were 2 of detachment of the retina, both giving a positive complement fixation test and showing streptococcus viridans in culture; 2 of chronic iridocyclitis, 1 having a positive and 1 a negative complement fixation test, but both showing streptococcus viridans in culture; 1 of choroiditis with a negative complement fixation test and showing streptococcus, staphylococcus, bacillus pyocyaneus, and pneumococcus in culture, and 1 of retinal hemorrhage, in which the complement fixation test was not made, and showing streptococcus and staphylococcus in culture.

Besides the elimination of the dental focus the patients received the usual treatment indicated in the various conditions, such as, atropine, leeching, sodium salicylate, etc. Of the 53 cases, 7 or 14 + % were cured; 27 or 50 + % were improved, and in 19 or 35 + % there was no improvement. 24 of the 53 cases were of an acute nature and 29 were of a chronic nature. As might be expected, the cases which

showed the most benefit as the result of dental treatment were acute conditions, while the cases in which no improvement was noted were mostly of a chronic nature. Of the 24 acute cases 4 or 16 + % were cured; 18 or 75% were improved, and in 2 or 8 + % there was no improvement. Of the 29 cases of a chronic nature, 3 or 10 + % were cured; 9 or 30 + % were improved, and in 17 or 58 + % there was no improvement.

In summarizing the results of the investigation the following conclusions are reached:

(1) Dental infection does not present a definite clinical entity, although it is most often seen in the form of a low-grade chronic infection involving the iris, choroid, and ciliary body.

(2) Dental infection is more frequent than has heretofore been believed and it must be carefully sought for by means of the X-ray.

(3) The infection is carried to the eye in all probability through the lymphatics and osseous channels.

(4) The streptococcus viridans is the organism most frequently found in cultures from tooth roots or sockets and is the main bacteriological factor. Other organisms, as the staphylococcus, pneumococcus, and bacillus pyocyaneus, must also be considered.

(5) In acute cases a mixed infection is the rule, the streptococcus viridans being most often associated with the staphylococcus; whereas in chronic cases the viridans alone is common.

(6) The streptococcus complement fixation test has not given satisfactory results, the findings not being consistent with the clinical picture.

(7) Vaccine treatment has been disappointing. The percentage of cures in cases receiving vaccines was only 11 + % as compared to a general percentage of cures of 14 + % of the cases treated. The administration of vaccines in acute cases gave better results than in chronic cases and those cases presenting a positive streptococcus complement fixation test with streptococcus viridans in the culture seemed to be benefited more by the administration of vaccines than others.

(8) The factors most concerned in the cure or improvement of these cases were the extraction of the offending teeth and the resulting drainage of the infected sockets.

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EYE MANIFESTATIONS IN A CASE OF POLYCYTHÆMIA.

By Dr. MARTIN COHEN, New York.

MRS. E. J., 60 years of age, family history irrelevant. In the past history, one spontaneous miscarriage, and deafness in the left ear for the past three years are the only noteworthy facts.

The condition reported dates back two years, when she first came under observation at the Post-Graduate Hospital. At that time she complained of failing vision in the right eye, hot flushes, and other symptoms of polycythæmia, and also manifestations of an arteriosclerotic involvement of the kidney.

Physical examination showed plethora and cyanosis of the skin and mucous membrane, a moderate degree of emphysema, a normal cardiac condition, and an enlarged spleen and liver. There were no other glandular changes.

Blood pressure, slightly increased; the systolic, 175; diastolic, 85.

The urine was scanty, 300 to 900*cc*, and of low specific gravity; there were a few granular casts and a trace of albumin, characteristic of a chronic interstitial nephritis.

The phenosulphothalein test was about normal (40 per cent.). The blood contained between 8 and 10 million red blood cells, which is double the normal quantity, and a hæmoglobin of 140 to 160 per cent. The white blood cells were relatively normal, except for a slight increase of lymphocytes; there were no abnormalities of the other elements or blood platelets; the coagulation and bleeding time were normal. The viscosity test was not determined. The chemical examination indicated no acidosis.

A finding of considerable importance was a strongly positive Wassermann blood test. The treatment consisted mainly of venesection of 900*cc* of blood, followed by an infusion of 500*cc* of saline solution; this treatment was

repeated after a few weeks and resulted in a lessening of the degree of cyanosis and an improvement in the general symptoms, including the venous hyperæmia of the retinal vessels.

The examination of the fundi two years ago showed a thrombosis of the right central retinal vein, probably not due to the mild nephritis or the mild arteriosclerosis, and a fundus polycythæmicus of both eyes. At present, the ocular examination is as follows:

Right eye, no light perception; pupil slightly dilated, rendering no response to direct examination but giving an indirect light reaction; no abnormal deviation of the eyeball; the intraocular tension is normal; the tarsal conjunctivæ and caruncles are hyperæmic; the papilla is of a steel gray color, its nasal border alone is moderately blurred; the macular region is studded with a few ill-defined grayish and pigmented spots.

The vascular system is practically similar in both eyes. The retinal veins are characteristic of the polycythæmic condition; they are markedly dilated, their distension extending far into the periphery of the fundus; they are also uneven in caliber, have a distinctly bluish appearance, and several U-shaped indentations of the entire vessel wall occur along their course,—which are independent of the arterial compression of the veins; along one of their borders and at some distance from the disk, the superior and inferior retinal veins are surrounded by whitish streaks which are probably due to a lymphstasis. Venous compression due to arterial crossing is observed over a few places of the fundus. The arteries are apparently increased in number on and near the disk, which is due to an injection of the normal and invisible branches; they are also tortuous, normal in caliber, and their central light reflex streak is prominent.

Left disk: Its color is yellowish white on the temporal side, with a sharply outlined margin, but the nasal side is elevated to three diopters. Fundus findings: fundus polycythæmicus, R. and L.; retinal arteriosclerosis R. and L.; post-neuritic optic atrophy and central chorioretinitis R., and papillitis in left eye. Although one would expect to find a retinal cyanosis in polycythæmia, still this case is of interest for the following reasons:

1. The association of lues with polycythæmia is rare; still, in this case the combination might be coincidental.

2. The presence of a strongly positive Wassermann reaction, a bilateral retinal arteriosclerosis, and a unilateral central

chorioretinitis suggests the presence of a specific retinal arteriosclerosis.

3. Thrombosis as a complication of polycythæmia has been described, but its location in the central retinal vein in this condition has not, to my knowledge, been previously recorded in the literature.

4. At a recent section meeting I reported a case which demonstrated the increase of the white blood cells in the blood with leukæmic manifestations in the retina and choroid; this case is almost a counterpart, showing increase of the red blood cells in the blood and characteristic polycythæmic manifestations in the retina.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

An ordinary meeting of the Section was held on Wednesday, December 12, 1917, Mr. William Lang, F.R.C.S., the President of the Section, being in the Chair.

Disturbances of Vision by Cerebral Lesions.

The whole sitting was devoted to the reception of an important contribution on this subject by Lieut.-Colonel Gordon Holmes, M.D., who illustrated his thesis by means of many photographs and diagrams. It followed the contribution, eighteen months ago, of the paper by Colonel Lister and the present author, before the same body, on the disturbances of vision produced by gunshot injuries of the visual cortex and of the optic radiations. Certain conclusions on the cortical representation of the retina were evolved, especially the segmental correspondence of different areas of the retina with separate zones of the cortical visual area. The main conclusions set out in the former paper were:

1. The upper half of each retina is represented in the dorsal, the lower in the ventral part of each visual area.

2. The center for macular or central vision lies in the posterior extremities of the visual areas, probably in the margins and the lateral surfaces of the occipital poles. The macular region has not a bilateral representation.

3. The center for vision subserved by the periphery of the retina is probably situated in the anterior end of the visual

area, and the serial concentric zones of the retina from the macula to the periphery are probably represented in this order from behind forwards in the visual area.

The foregoing agree with those already arrived at by Inouye from a study of cases in the Russo-Japanese war, and by others. The author has now seen a large number of cases since the last communication to the Section, and all the observations on these harmonized with the foregoing conclusions.

Central and paracentral scotomata are very common in all moderate injuries of the occipital lobes. Injury of the poles of both hemispheres may cause complete loss of central vision; an unilateral wound produces homonymous scotomata in the opposite halves of the fields. A common type of case is that of pure lateral paracentral scotomata, of which an instance was that of a man whose helmet was penetrated by a shrapnel ball, rendering him quite blind until next day. No subjective visual phenomena were noticed. From a small penetrating wound, softened brain tissue extruded, just to the right of the mid-line, and one inch above the inion. The radiograph showed much depressed bone, but no foreign body. After the removal of brain tissue and bone from the wound, the sight was quickly recovered. A week later, there was a large left homonymous paracentral scotoma, but peripheral vision was unaffected and the color fields were normal in the right halves, but there was no perception of either red or green to the left of the middle line.

In all patients with inferior unilateral paracentral scotomata the injury involved the tip or posterior portion of one occipital lobe, at or immediately above the level of the calcarine fissure, and usually the size of the scotoma had a relation to the depth of the wound. Some cases of bilateral inferior scotomata afford valuable evidence on the subject of cortical localization. Isolated superior paracentral scotomata are much less common, and in the few cases seen by various people the lesion appeared to involve the lower part of the area striata: indicating, therefore, that the retina immediately below the macula is represented in the lower portion of the calcarine area. There seems to be sufficient evidence to allow the conclusion to be formed that the upper halves of the retina are projected on to the

dorsal part of the visual area, and the lower halves on to its ventral portion. Macular and perimacular vision are represented in the most posterior part of this area. Still, other problems require consideration, and they can best be studied in cases with paracentral scotomata. Usually, the sector scotomata lie between the vertical line through the fixation-point and one of the oblique radii.

The author then proceeded to deal with the cortical representation of peripheral vision. In the previous communication, Colonel Lister and he could only suggest, by a process of exclusion, that vision in the periphery of the fields was represented in the anterior portion of the cortical visual area. It was not to be expected that many cases suitable for giving definite evidence on this would be seen, as perforating wounds which could involve the anterior portion of the area striata would very likely involve the optic radiations also, and so cause intensive or irregular areas of blindness. A recent case which came under Riddock's observation, however, gives valuable positive evidence of the localization of peripheral vision. This man had considerable peripheral contraction of both visual fields. A rifle bullet and, later, fragments of bone were removed from the great longitudinal fissure between the two hemispheres, at such a level and depth that the anterior portion of the calcarine area was probably injured. The author himself had recently seen a case which was valuable from the point of view of the elucidation of this question, and he narrated it in detail. The cases seen afforded some positive evidence that peripheral vision is represented in the anterior portions of the calcarine area. When these observations are associated with the fact that peripheral vision is never affected by local lesions of the posterior part of this area, the hypothesis becomes very probable if it be admitted that the visual sphere at least is roughly approximate with the area striata.

Destructive injuries of the optic radiations generally produce a complete hemianopia, in which the blindness may or may not reach the fixation point, or large irregular areas of blindness. Occasionally the visual defect is more regular, and these affect the important question whether the fibers of the radiations which carry impressions from the retina and

end in definite portions of the visual cortex, are arranged in order according to the origin of the impressions they carry and to their exact termination. The author has seen a number of cases in which quadrantic hemianopias or other defects of the fields resulted from wounds of the radiations; some of these he quoted. The author was forced to the conclusion that the fibers of the radiations are arranged regularly in laminæ or series, according to the impulses they carry. With regard to the course in the radiations of the fibers concerned with macular vision, observations suggest that these have a distinct path of their own, but he had not yet seen a case in which a paracentral scotoma, or an isolated affection of central or pericentral vision could be attributed to a lesion of the radiations.

Colonel Holmes then discussed the nature of the lesions. With regard to concussion, in many of his patients the defects remained unaltered at repeated examinations, though these examinations were extended over considerable periods. In others, however, there was a progressive diminution of the blind area, sometimes rapid, at other times slow. In such a highly-organized function as that of sight, recovery can hardly ever be due to vicarious representation or to the assumption of the function of the destroyed cortical areas by other centers. When the defects persist for a considerable time after the infliction of the wound, he did not doubt they depend on a simple destructive lesion. But in the earlier stages of gunshot injuries of the head it is not uncommon to find a complete hemianopia or a large area of total blindness disappear in a few days or weeks, probably by recovery from the center to the periphery. But peripheral vision often reappears before the central or pericentral. He attributed the condition when recovery was quick to an œdematous swelling of the areas concerned, analogous to that which occurs in the myelin sheaths of the spinal cord. He could not say whether a molecular disturbance might contribute to the defect. The rapidity of recovery argued a condition short of the destruction of anatomical integrity. It had been common to find a zone of partial vision around the scotomata, or on the borders of a quadrantic or incomplete hemianopia. But in most of the cases examined more than three weeks after the onset of the

blindness the margins of the scotomata were remarkably sharp. He passed on to discuss the mechanism and function of peripheral vision, and thence considered the disturbances of color perception caused by cerebral lesions. He said it had not been conclusively shown that color perception may be completely lost in any part of the field when that of light or white is undisturbed; many of the hemiachromatopsias have been instances of hemiamblyopia. He had frequently observed among his cases that red and green test-objects could not be recognized in certain regions, often in homonymous halves of the visual fields, but in every instance visual sensibility to white test-objects of the same size was reduced. His observations led him to the conclusion that an isolated loss or dissociation of color vision is not produced by cerebral lesions.

The paper concluded with a discussion of disturbances of visual attention, and loss of visual orientation and appreciation of depth. He summarized the conclusions arrived at—which, he said, could at least serve as a working hypothesis for further investigations—in the following way:

1. The upper half of each retina is represented in the dorsal, and the lower half in the ventral part of each visual area.

2. The center for macular or central vision lies in the most posterior part of the visual areas, probably on the margins and in the lateral surfaces of the occipital poles. The macula has not a bilateral representation.

3. The center for vision subserved by the periphery of the retinæ is situated in the anterior portions of the visual areas, and the serial concentric zones of the retinæ from the macula to the periphery are probably represented in this order from behind forwards in the visual areas.

4. Those portions of the retina adjoining their vertical axes are probably represented in dorsal and ventral margins of the visual areas, while that in the neighborhood of the horizontal axes is projected on to the walls and the floor of the calcarine fissures.

5. Severe lesions of the visual cortex produce complete blindness in the corresponding portions of the visual fields, or, if incomplete, an amblyopia, color vision being generally

lost, and white objects appearing indistinct; or only more potent stimuli, such as objects moving sharply, may excite sensations.

6. The defects of vision in the fields of the two eyes are always congruous and superimposable, provided that no disease or injury of the peripheral visual apparatus exists.

7. Lesions of the lateral surfaces of the hemispheres, particularly of the posterior parietal regions, may cause certain disturbances of the higher visual perceptual functions with intact visual sensibility, as loss of visual orientation and localization in space, disturbance of the perception of depth and distance, visual attention loss, and visual agnosia.

On the motion of Dr. JAMES TAYLOR, past-president of the Section of Neurology, the author was the recipient of a special vote of thanks for his contribution.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By LEWIS W. CRIGLER, M.D., Secretary.

OCTOBER 15, 1917. GEORGE H. BELL, M.D., CHAIRMAN.

Dr. J. Bruder presented a patient suffering with a **gumma of left lower lid.** Man, age 34, gave a history of having contracted syphilis twelve years previously. Two months ago he noticed a swelling of left lower lid which lasted for a few days and then became ulcerated. When first seen by Dr. Bruder a month ago, he presented the following condition:

An irregular, eroded, and punched out ulcer near the outer canthus and extending to the palpebral margin. The preauricular gland was swollen. The floor of the ulcer was covered with a dirty gray exudate. The conjunctiva in the region of the wound was slightly thickened. Wassermann reaction, ++++ positive.

Discussion: Dr. A. E. Davis called attention to the fact that malignant growths of the lid simulate, clinically, gummas, and that a thorough physical and microscopical examination of the lesion is often essential.

Dr. Heller spoke of an accidental inoculation of the lid from a vaccination pustule on the arm of a patient which showed some of the characteristics of a gumma. Dr. Bruder said that a more general use of the Wassermann reaction, in patients presenting various syphilitic stigmata, should be performed as a prophylactic measure against future luetic involvements.

Dr. J. Bruder presented a case of **keratitis profunda.** Patient, female, age 24, was first seen by Dr. Bruder in June,

1917. She presented the following symptoms: sudden attack of pain and obscuration of vision in left eye; severe frontal headache on left side. Her vision was reduced to hand movements, the right remaining normal. Tension normal in affected eye. Examination with loupe revealed a diffuse uniform gray opacity, situated in the center of the cornea. The opacity is seen to be composed of numerous maculæ and interlacing striæ with an ill-defined border. Inflammatory symptoms were rather mild, while the irritative symptoms were quite marked. After a few days the opacity spread over the cornea, leaving only a narrow rim inside the limbus which remained clear. No deep vessels were present. Instillation of fluorescein produced a deep stain of the cornea. Wassermann and tuberculin tests were negative. X-ray examination of the teeth showed disease of left upper and second lower bicuspid, at the apices. The sinuses were normal.

DISCUSSION: Dr. REIDEL was of the opinion that thyroid-extract treatment should be tried in this case in view of the negative clinical findings.

Dr. H. H. Tyson stated that one year ago he had under treatment an eye, similar to Dr. Bruder's case, in a robust Irishman, in which all causes could be excluded except his teeth. He had a severe case of pyorrhœa alveolaris but would not consult a dentist or receive any treatment for his mouth or teeth. Notwithstanding this fact, under local ocular treatment and with Fowler's solution internally, the keratitis which lasted six months finally disappeared, leaving him with practically normal vision. The interesting point was that in spite of the continued presence of the supposed cause, the eye completely recovered.

Dr. GEO. H. BELL referred to the examination of the teeth, tonsils, and gastro-intestinal tract as a source of focal infection in these cases, when the usual physical examinations were negative, and also reported satisfactory results in a few cases when treated along these specific lines. He said that in as much as the ætiology of this rare condition was so obscure, it was important to avail one's self of all the means of diagnosis obtainable.

Dr. BRUDER presented three cases of **retinitis pigmentosa.**

Dr. GEO. H. BELL presented a case of **senile keratosis of the**

upper lid. Patient came to his clinic at the Infirmary with a thickened upper lid which looked like thickened epidermic patches. The elevations felt rough and horny. The patient was referred to the Skin and Cancer Hospital for diagnosis. It was there pronounced **senile keratosis, secondary to xanthoma.**

The prognosis is that it may remain quiescent or later undergo epitheliomatous degeneration. An ointment of ammoniated mercury was prescribed.

Dr. MARTIN COHEN presented a case of **polycythæmia with eye manifestations.** Mrs. E. J., age 60 years, has a mild form of chronic interstitial nephritis, arteriosclerosis, enlargement of the liver and spleen, a strongly positive Wassermann reaction, and a typical blood picture and physical signs of polycythæmia. Although one would expect to find similar changes in the eye as in the general condition in polycythæmia, still this case is of interest for the following reasons:

First, the association of polycythæmia with lues.

Second, the presence of a specific retinal arteriosclerosis.

Third, thrombosis as a complication of polycythæmia has been described, but not its location in the central retinal vein. Thrombosis of the central veins occurs in nephritis or arteriosclerosis or both, but these conditions were of a mild type in this patient, and therefore would not likely result in the fundus findings as mentioned.

DISCUSSION: Dr. TYSON in discussing Dr. Cohen's case took exception to his statement that the appearance of the eye backgrounds in his case was pathognomonic of polycythæmia. He considered it a case of angiosclerosis of the retinal vessels and that it was the same picture often presented in senile angiosclerosis. He did not doubt but that polycythæmia existed in the case, but did not agree with Dr. Cohen that it was the cause of the ocular condition. He said that similar cases had been reported by Oatman and others in which no polycythæmia existed.

Dr. WOOTTON remarked that the case represented an angiopathic sclerosis, but that he was not acquainted with the appearance of fundi in polycythæmia.

Dr. COHEN stated that there were undoubtedly angiosclerotic changes in the retinæ, but that the association of polycy-

thæmia caused typical changes in the veins which were pathognomonic of polycythæmia.

Dr. H. H. TYSON reported a case of **traumatic paralysis of the left abducens nerve,** caused by injury following a fall.

History: Lieut. E. C. B., Royal Flying Corps, age 23 years, while flying at an altitude of 400 feet, on Sept. 13, 1917, was struck by another plane, both planes falling to the ground. Two officers were killed, Lieut. B. escaping with a fractured clavicle and nose and extensive orbital, subconjunctival, and subcutaneous hemorrhages, with a contused wound of left brow. On Sept. 21, 1917, vision each eye $\frac{20}{20}$; had homonymous diplopia in his entire left field and extending slightly into his right field. Pain on efforts at extreme rotation to right or left. X-ray negative, fundus, tension, and vision normal. On Sept. 27th single vision existed in entire right field and ten degrees to left of median line. Oct. 10th, diplopia begins at 20 degrees to left of median line; O. S. abduction 35°—adduction, 50°; O. D. abduction 45°, adduction 55°. Pain in left eye on attempts at extreme rotation. Pupils equal, reactions normal. No ptosis. Still moderate amount of subconjunctival hemorrhage is present four weeks after date of injury. The paralysis of the abducens in this case is evidently caused by pressure, and on account of the long course traveled by the nerve, it would be difficult to state the exact site of the injury, but considering the steady improvement in motility coincident with the disappearance of the hemorrhage it is safe to assume that it is of orbital origin and that the prognosis is good. The history of similar traumatic cases reported is that they require from six to twelve weeks for complete recovery, while there are a few which recover only partially. Sodium iodide was given internally.

DISCUSSION: Dr. LESZYNSKY considered the cause as due to a nuclear hemorrhage on account of the involvement of an isolated muscle and with no evidence of fracture.

Dr. COHEN was of the opinion that the paralysis was due to a partial severing of the nerve and not a hemorrhage in the orbit or nuclear center of the sixth nerve.

NOVEMBER 19, 1917.

Dr. BEN WITT KEY presented a case of **extensive penetrating**

wound of the globe. The case was that of a boy 19 years of age who had been struck in the left eye by the end of a wire hoop eleven days before presenting himself for treatment. The cornea had been penetrated from the outer limbus directly across to the nasal limbus in a horizontal line just above the pupillary margin and extending into the sclerotic at the nasal side for about *2mm*. The line of perforation measured 11*mm*. The iris was prolapsed in the nasal third of the wound and incarcerated with the exception of about *2mm* of the lower margin. The iris was freed through wound dissection, iridectomy, and replacement. A conjunctival flap was then made covering the wound and recovery was uneventful, leaving a scar which is covered by the upper lid and a moderate amount of astigmatism. Vision with correcting lenses was $\frac{20}{30}+$ when last examined.

DISCUSSION: Dr. CRIGLER said that several years ago he had a case very similar to the case presented by Dr. Key: A young man, while playing handball with his myopic glasses on, received a blow which struck the right glass. The glass broke and was driven into the eye. A triangular cut was made in the cornea, one prong of which extended horizontally through the sclera several *mm* behind the ciliary body. There was escape of both aqueous and vitreous on removal of the piece of glass. The prolapsed iris was cut off and pillars replaced. There was no prolapse of ciliary body or choroid. The scleral wound was closed by conjunctival sutures only, with the exception of one stitch placed through the episcleral tissues at the limbus. Both eyes were bandaged. Recovery was rapid without the least signs of inflammation, and the remarkable feature in the case is, that, although both the anterior and vitreous chambers were opened the lens was not injured. Vision was restored to $\frac{20}{40}+$ with correcting lenses.

Dr. KEY presented a case of **extensive ciliary staphyloma** in a man 62 years of age. He gave a history of having been struck in the right eye forty years ago. Since that time the eye has been blind but there have been periodic attacks of pain. Five months ago the eye became very painful and began to "swell." The appearance now is that of almost complete ciliary staphyloma. The cornea is infiltrated with calcareous deposits. Dr. Key has advised that the eye be removed.

Dr. S. T. HUBBARD presented a case of **optic neuritis of accessory sinus origin followed by complete recovery** after opening and draining these cavities. Patient, female, age 25, was seen for the first time in April, 1916. Her symptoms then were pain and tenderness of the left eye aggravated by movement, intense headache, dizziness in walking, and marked blurring of her vision. Her previous history was that of a pus appendix and a suppurating ear. Her sight grew steadily worse and at the end of a week from onset her direct vision was lost completely, with only blurred peripheral vision remaining. With this latter symptom she suffered with intense headache which was aggravated by coughing.

Examination of the backgrounds at this time revealed the right eye to be practically normal; vision $\frac{20}{20}$. The left eye showed a swelling of the disk with small hemorrhages present. There were also hemorrhages in the adjacent retina on the temporal side. There was a relative central scotoma. X-ray by Dr. Dixon revealed rather doubtful signs of active inflammation although there was some haziness in the frontals and sphenoids. Vision at this time was reduced to fingers at eighteen inches.

The patient was referred to Dr. Craig who opened and thoroughly drained the accessory sinuses involved. Within two weeks from date of operation the retinal œdema and hemorrhages had disappeared and the patient's vision restored to practically normal.

One year after the trouble in the left eye, the same condition developed in the right eye. It was treated in a similar manner as the left with like results. At the present time there is a contraction of the fields for color and an enlargement of the normal blind spot in both eyes.

Dr. CHARLES MACK presented a case of **hole in the retina at the macula.** The case presented no unusual features. The patient gave a history of having been struck in the right eye with a cork six years previously. There is a central scotoma of 10°.

DISCUSSION: Dr. WOOTTON stated that after the lapse of years these holes sometimes become surrounded by colloid dots.

Dr. COHEN referred to cases that had been reported of idiopathic origin in which the lesion was bilateral.

Dr. J. H. CLAIBORNE presented a case of **glaucoma success-**

fully operated on by iridectomy for the object of bringing about a discussion on the latest and best mode of procedure in the treatment.

DISCUSSION: Dr. WOLFF said that iridectomy was still the operation of choice in the treatment of acute glaucoma, but that it was better to control the initial attack by the aid of miotics if possible and then perform iridectomy at the more favorable time when the congestion had subsided and the anterior chamber returned to more nearly its normal depth. Failing to reduce the tension with miotics iridectomy should be resorted to at once, preceded by posterior sclerotomy.

Dr. BELL does not think well of posterior sclerotomy as a preliminary step in the performance of iridectomy for the relief of acute glaucoma because of the risk of vitreous hemorrhage, infection, and subsequent detachment of the retina. Dr. Bell said that he would not be surprised that we would some day find that the toxines produced by abscessed teeth, diseased tonsils, and disorders in the gastro-intestinal tract are responsible for a great many of the cases of glaucoma.

Dr. BRUDER referred to a case of glaucoma that had come under his observation in which the tension was subnormal. A few hours after the instillation of atropin it rose above normal and continued so until the effect of the atropin had been overcome.

Dr. H. W. WOOTTON presented a patient with unusual **retinal detachment.** The case was that of a man, 32 years of age, without history of trauma or constitutional disease. For the past six months he has complained of disturbed vision in the right eye. Previous to that he said that he had been disturbed for a long time by what appeared to be floating clouds before the field of vision of this eye. When first seen vision was reduced to fingers at two feet; field of vision contracted to the upper and outer quadrant. The ophthalmoscopic picture was that of a flat wavy membrane resembling a retina that had been torn from the ora serrata and extending from above downward in an oblique direction in the temporal half of the pupillary space. In as much as the fundus showed a normal red reflex to the outer side of the membrane there was a question as to whether or not the mass was a proliferating retinitis.

Dr. WOLFF thought that the retina had been torn from the temporal side.

Dr. BRUDER said that he could see vessels on both sides of the membrane and that therefore it must be a proliferating retinitis.

In Dr. BELL's opinion the retina was torn and folded upon itself.

Dr. WOOTTON presented a case of **retinitis proliferans in a diabetic subject.** In addition to the diabetes there was also a chronic interstitial nephritis and arteriosclerosis. The patient, 53 years of age, has been under treatment for diabetes for the past five years. Her vision began to fail in the left eye five months ago. Until that time she was not aware that vision in her right eye was defective. When first seen by Dr. Wootton her vision was reduced to light perception Fundus examination revealed a grayish white mass at the site of the disk, protruding *2mm* into the vitreous, somewhat larger than the disk in diameter. The left eye presented a typical picture of diabetic retinitis.

Dr. Wootton interpreted the picture in the right eye as a proliferating retinitis of a high degree involving mainly the papilla. The surface appeared uneven and the margin irregular, due to prolongation of connective tissue. The veins were enlarged and tortuous and the arteries contracted. Two small hemorrhages bordered the upper surface.

DISCUSSION: Dr. COHEN said that the case was unquestionably one of proliferating retinitis following a hemorrhage. He said that thrombosis of the central vein usually resulted in optic atrophy. In this case atrophy does not exist.

Dr. TENNER asked the speaker if he did not consider retinitis proliferans always due to hemorrhage. Dr. WOOTTON replied that he did.

Dr. F. N. IRWIN presented an unusual case of **scleritis, of possible tuberculous origin.** The present attack has existed for one month. Patient states that several years ago the same eye had been inflamed and she was then given injections. The eye had also been inflamed in infancy. The Wassermann reaction is negative, likewise Von Pirquet. Dr. Irwin has removed a piece of the tissue and injected it into a rabbit. He will report results at the next meeting.

DISCUSSION: Dr. SHERMAN said that he had had several cases of this type which he had treated with tuberculin. He was skeptical as regards its virtue but the good results obtained by others convinced him of its value.

Dr. GEORGE H. BELL showed a case of **sarcoma of the orbit** and exhibited X-ray plates of same. The case was far advanced and no operative interference was advised. He proposes to try radium.

Dr. BELL also presented a case of **plastic iritis** in both eyes due to severe mouth infection. When first seen both pupils were contracted and the aqueous cloudy. There was severe pain in both eyes, considerable exudation in the left pupillary space and marked ciliary injection. Wassermann test was +++ positive. The usual treatment for iritis was begun, including daily inunctions of mercury. In as much as the patient's teeth were badly decayed, fourteen were extracted during his first seven days' treatment at the hospital. Improvement in his eye condition was so rapid that Dr. Bell decided to discontinue the mercurial inunctions. The patient made a rapid recovery, leaving the hospital at the end of three weeks. He has now, slightly over nine weeks since treatment was begun, gained fourteen pounds in weight, his eyes are perfectly quiet, and his vision is $\frac{20}{20}$ in each eye. He was given only seven inunctions of mercury. Dr. Bell thinks that unquestionably the condition of the teeth in this case was responsible for the iritis. He proposes to treat the patient for constitutional syphilis later.

DISCUSSION: Dr. SCHOENBERG said that the case was a very interesting illustration of how difficult it is at times to make a diagnosis of a non-syphilitic condition in a patient with syphilis. If the treatment had consisted only of the extraction of the teeth Dr. Bell's diagnosis could not have been disputed. But this patient received several inunctions of mercury before his decayed teeth were removed. The fact that the iritis began to improve after the mercurial inunctions were discontinued does not prove anything, since it is a well established fact that mercury, when absorbed, remains in the system for months and even years, during which time it continues its effect.

Dr. DWYER said that it is possible to recognize different

types of iritis clinically and that he had no doubt but that Dr. Bell's case was one due to mouth infection. He said that recovery was too rapid in this case for it to be classified as syphilitic.

Dr. HUBBARD referred to a case of **kerato-iritis** which recovered very rapidly after the extraction of several offending teeth.

Dr. ALGER thinks we may be too optimistic in regard to this new discovery but reported the case of a man who had had more than 100 attacks of iritis during the past thirty years. He had had every form of examination and treatment without result. During the last attack, two years ago, his teeth were found to be bad, those diseased were removed, the iritis subsided much more quickly than ever before, and there has been no return since.

Dr. BELL said that, according to his observation, it was not reasonable to assume that seven inunctions of mercury would cure a plastic iritis of specific origin.

Dr. BEN WITT KEY reported a case of **pulsating exophthalmos.** J. O., aged 49, admitted to the hospital Oct. 26, 1917, with history of a severe blow with a club on right side of head two months before. Five days after the injury he noticed slight protrusion of his left eye, which has gradually become worse; he is also conscious of a pulsation and noise on left side of his head. On inspection a definite pulsation of the globe synchronous with each heart pulsation could be seen. A well-defined bruit could be heard. X-ray plate gave no evidence of injury. Digital pressure caused the pulsation to stop. At the end of two weeks the patient was discharged from the hospital, the exophthalmos being greatly diminished and the pulsation very feeble.

BOOK REVIEWS

I.—**The Indian Operation of Couching for Cataract.** Incorporating the Hunterian Lectures, Feb. 19 and 21, 1917. By Lieut.-Col. R. H. ELLIOT. With 45 illustrations, pp. 94. H. K. Lewis, London, 1917. 7/6 net.

The couching of cataract is one of the oldest operations in surgery which has gradually given way to better procedures, except in India where its popularity continues, particularly owing to social and religious conditions, though thanks to the efficient members of the I. M. S. the newer procedures are gaining steadily. As the operation of couching is still recommended under exceptional conditions by well-known operators in the West, Colonel Elliot's book arouses practical as well as historical interest. The author distinguishes between two methods of couching, depression and reclination; in the first the lens is pushed straight into the vitreous, while in the second, the later method dating from 1785, the lens is reclined backward, remaining attached by the suspensory ligament below. The site of approach is either anterior or posterior, as the puncture is made with a needle-like instrument through the aqueous chamber or posterior to the ciliary body.

The results of this operation as practiced by the native coucher, Colonel Elliot, from a study of many cases (780), finds to be as follows: quite a number of eyes are lost from panophthalmitis and iridocyclitis. Cases were usually seen from 1 to 10 years following the couching. The results are universally bad, 60% are lost. The vision in only 10% was $\frac{1}{2}$ or better when the patients came under observation, the shorter the period after operation the better the sight. The causes of failure in the total cases were iridocyclitis 35%,

glaucoma 11%, imperfect dislocation of lens 9%, retina detached 1.5%, optic atrophy 2.5%, retinal pigmentation 0.5%, chorioretinitis 1.1½%. The iridocyclitis and glaucoma generally come on within a few days of operation. Detachment of the retina probably occurs in a greater percentage. As for the question whether the lens in the vitreous brings about retinal changes, as has been advanced by some, Elliot cannot find evidence to support this view, though he has found an undue distinctness of the choroidal vessels.

Fifty-four globes were available for microscopic examination. The cataractous lens was usually found floating freely in the vitreous or it was bound down to the ciliary body by exudate. In four the lens was displaced into the anterior chamber. The eyes lost by panophthalmitis were eviscerated and not available for examination. The evidences of iridocyclitis were frequent and plastic in character. The author's observation on changes in the vitreous are important. The vitreous was found detached and shrunken, with evidences of infiltration and of organization. The vitreous exudates under the microscope proved to be of the nature of inflammatory exudation, more or less structureless masses containing blood-cells and leucocytes; the anterior hyaloid membrane was definitely thickened and infiltrated. The optic nerve-head was usually inflamed and the author regards this as proving the existence of a lymph current in the vitreous body. Detachment of the retina occurred in 70% of these eyeballs, undoubtedly secondary to the vitreous changes. Glaucoma was present in nineteen eyeballs, the cause was always one of those accepted for secondary glaucoma.

The diagnosis of a couched cataract is next taken up and the question of what is to be done with these cases comes up for consideration. As a rule operators shrink from operating on these cases, and Elliot would urge that only the freely movable cataracts should be touched. In 1912 the author reported on the results of 18 cases in which the lens was removed; in 12 vision was improved, in 4 no change, and 2 were made worse, though he fears that the improvement was not lasting. It is fortunate for us that Colonel Elliot was interested in this remarkable operation and took advantage of the opportunities in India of collecting material and obser-

vations to write this complete and scientific study of the subject. While we in the West rarely see the results of the couching of cataract, the future of eyes in which lenses have become dislocated into the vitreous is important.

A. K.

II.—**Congenital Word-Blindness.** By Dr. JAMES HINSHELWOOD, Glasgow, pp. 112, published by H. K. Lewis & Co., Ltd., London, 1917. Price 4/net.

The studies and observations of the writer on word blindness, a subject to which he was the first to draw attention, are now collected in one volume which will be of great service to those dealing with congenital defects of childhood, particularly from its educational side. Word blindness means that with normal vision for letters and words the patient cannot interpret printed or written language.

In the first chapter the subject of acquired word-blindness is reviewed, as a knowledge of this condition is necessary to explain the congenital condition. The case history of a patient is reported whom the author was able to follow for many years after the onset of the cerebral lesion, and on whom an autopsy was obtained. This patient lost his visual memory for printed characters though he was able to read numbers and write to dictation. The only other cerebral defect was a right lateral homonymous hemianopsia. At autopsy a subcortical lesion was found causing isolation of the visual memory center, leaving this center intact.

The place where visual impressions are stored is the visual memory center, situated in the angular gyrus on the left side in right-handed people. Visual memory, the author has shown, can be furthermore subdivided into functions whose loss produce letter, word, and mind blindness. It is also to be remembered that there are independent visual memories for words and letters, for numbers and for musical notes, of which a single group alone can be lost. While these require mental attention and concentration, the author speaks of another group of visual memory for objects, persons, places, etc., which may be stored up in the visual memory centres of both hemispheres. Cases have been reported showing that there are partial forms of word blindness where an individual becomes

word blind to only one language. This suggests that our visual impressions are arranged in definite and ordered groups.

The visual memory center in the left angular convolution is connected with both occipital visual centers. If the visual memory center is destroyed by a cortical lesion, the word-blindness is accompanied by agraphia; a subcortical lesion, on the other hand, leaves the center isolated and cut off from other centers and communicating fibers, but the power to write is retained.

Cases of acquired word-blindness occasionally recover. When this occurs, improvement begins early. In those cases where the condition is permanent, something can be done by re-education. Hinshelwood believes that in these cases the corresponding center on the opposite side of the brain is educated, a procedure which has been noted in aphasia.

Congenital word-blindness is a defect in otherwise normal children, characterized by difficulty in learning to read, though they do remarkably well in oral instruction. The condition in the author's opinion is by no means so rare, and its proper recognition is important, as these children are often erroneously neglected or punished.

Twelve cases are reported, all showing the same defect in ability to recognize print or writing but with all the other cerebral powers unaffected. In only three did the defect include the non-recognition of figures, and all of them learned to write. One case made good progress in music. Hinshelwood finds that this condition preponderates greatly in boys, —of the twelve, ten were males. Congenital word-blindness is sometimes hereditary. An instance is reported where of a family of eleven, four of the boys had the greatest difficulty to learn to read; in the succeeding generation two boys of an unaffected mother showed this peculiarity. In these cases the cause is regarded as defective development. The hemianopsia so frequently associated with acquired word-blindness due to disease is never met with in the congenital variety.

In diagnosis the author particularly insists on "the gravity of the defect but also on the purity of the symptoms." The cases must not be confounded with those of general failure of cerebral development. The general intelligence of these

children is quite up to the average, and their auditory memory is intact.

As to treatment, these children can be taught to read with proper treatment and great perseverance. The author recommends personal instruction alone, and not in a class, and the use of the old-fashioned method of learning to read as the best and quickest. We are all greatly indebted to Dr. Hinshelwood for his researches in this important subject, as cases with this defect are first brought to the oculist for examination and the diagnosis, as the author has well shown, is not difficult.

A. K.

III.—**Consumption and Squint**. One of three clinical studies in Tuberculous Predisposition. By W. C. RIVERS, M.R.C.S., L.R.C.P., D.P.H. Published by Geo. Allen & Unwin, London, 1917.

The author found six squinting consumptives in five hundred examined, or about 1.2 per cent. This seeming excessive, he examined nine hundred controls and found four who squinted, or about 0.4 per cent., from which he concluded that concomitant squint is two or three times as common in consumptives as in the general population. During the examination of 10,239 children (non-tubercular) Hansen found an average of 2.5 per cent. with strabismus, which shows a wide variation with the average of the author's controls.

As to the inferences one is entitled to draw as to a causal connection between strabismus and phthisis, he states that it is impossible to conceive how any simple ocular defect short of blindness could of itself favor the development of pulmonary tuberculosis, even in the most indirect manner. As the squint long preceded clinical manifestation of tuberculosis, it is unlikely to have been caused by that disease in any declared form. For the causal sequence to happen, necessary postulates are: early tuberculous infection, early interference with one single nervous function (the fusion faculty of vision or kindred function), and then complete latency for some—perhaps thirty—years. He says that squint appears to be sometimes a mark of mental deficiency or a tendency, personal or familial, to certain bodily anomalies or malformations. It probably instances the presence of predisposition to tubercle (clinical

pulmonary tubercle) without itself forming an active constituent of a phthisical diathesis. Such active constituent may be some associated malformation of which squint is the stigma. He finally concludes "that according to his findings on his evidence there is undoubted association between concomitant strabismus and consumption, of predisposition to which latter disease squint forms a sign."

While the author's observations are interesting, and it is conceded that strabismus may be associated with tuberculosis as with other diseases, many would hesitate to accept his deductions on account of the insufficiency of data, the omission of consideration of the individual ocular conditions, and the wide variance in his control ratios with those of other reliable observers.

H. H. T.

IV.—**Medical Ophthalmology.** By ARNOLD KNAPP, M.D. Being one volume of "An International System of Ophthalmic Practice," edited by WALTER L. PYLE, M.D. Philadelphia, P. Blakiston's Son & Co., 1918. Octavo, pp. 509, with 32 illustrations. Price $4.00.

There has been lacking hitherto a compact, systematic, well-indexed, single volume of reference in English devoted to the ocular symptoms of general medical diseases, hence the appearance of this book is very welcome.

The first section, consisting of 80 pages, written in collaboration with Prof. Marburg of Vienna, deals with the anatomy and physiology of the visual apparatus, and is illustrated with about thirty well-chosen diagrams. The next section, of 120 pages, treats of diseases of the nervous system, and following this come sections on diseases of the glands with internal secretion, diseases of the skin, circulation, and kidneys, digestive tracts, diseases of infectious nature and poisons.

The writer has drawn largely for his material upon the recognized German and French handbooks on these subjects and upon the transactions of the Ophthalmological Society of the United Kingdom. A captious reviewer might wish that he had exercised greater critical faculty in selecting and digesting some of his material, and that he had rejected some of the errors that are copied from one text-book to another. The

sections on syphilis, tuberculosis, and the nasal accessory cavities seem particularly valuable, since in them the writer's large personal experience has guided him to a great extent.

The volume will doubtless find its way into many hands and prove to be a very satisfactory book of reference.

W. A. H.

MISCELLANEOUS NOTES.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

PRESIDENT: E. TREACHER COLLINS, F.R.C.S.

ANNUAL CONGRESS, 1918.

The next **Annual Congress** of the Society will be held on **Thursday, Friday, and Saturday, 2d, 3d, and 4th of May, 1918.**

The arrangements will be as follows:

THURSDAY, MAY 2d.

(At the Royal Society of Medicine, 1 Wimpole Street, W.)

MORNING.

10 a. m. to 12.30 p.m.... Papers.

AFTERNOON.

2.30 p.m..... Discussion on "Plastic Surgery of the Eyelids." To be opened by Major H. D. GILLIES, Messrs. C. HIGGENS, and T. HARRISON BUTLER.

Members are invited to exhibit illustrative cases.

5 p.m..... Tea.

5.30 p.m..... Business Meeting.

Members will dine together in the evening.

FRIDAY, MAY 3d.

MORNING.	**Visit to the Metropolitan Asylums Board Ophthalmia School, Swanley.**
	Discussion on "Contagious Diseases of the Conjunctiva." To be opened by Messrs. J. B. STORY, SYDNEY STEPHENSON, Major J. F. CUNNINGHAM, and Capt. J. WHARTON.
AFTERNOON.	
3.30 p.m.	**Visit to Museum of Royal College of Surgeons.**
	Exhibition of Specimens illustrating War Injuries.
	Demonstration of Specimens of Eye Injuries by Col. W. T. LISTER.
EVENING.	**At the Royal Society of Medicine, 1 Wimpole Street, W.**
8 p.m.	Exhibition of Cases.
8.30 p.m.	Papers.

SATURDAY, MAY 4th.

MORNING.	**At the National Hospital for Paralysis, Queen's Square.**
10 a.m.	Clinical Meeting.
	Members are invited to show Ophthalmological Cases of Neurological Interest.

Members desirous of reading papers, showing cases, or taking part in the discussions, are requested to communicate as soon as possible with Mr. M. S. MAYOU, 30 Cavendish Square, London, W. 1.

Papers and communications, subject to the judgment of the Council, will be printed in full in the "Transactions."

Under the Bye-laws readers of papers must not exceed

twenty minutes, subsequent speakers ten minutes. The openers of the discussions are allowed twenty minutes.

All communications must be type-written.

A Museum will be held at the Royal Society of Medicine, with a Special Exhibition of Perimeters. Members wishing to exhibit drawings, specimens, or instruments should communicate with Mr. A. C. HUDSON, 50 Queen Anne Street, London, W. 1.

S. A. KINNIER WILSON, M. S. MAYOU } *Hon. Secs.*

THE AMERICAN JOURNAL OF OPHTHALMOLOGY

The first number of the new *American Journal of Ophthalmology* has been published, and the editors are to be complimented on its extremely creditable appearance. The journal is the result of the amalgamation of the *American Journal of Ophthalmology*, the *Annals of Ophthalmology*, *The Ophthalmic Record*, *Anales de Oftalmologia*, *Ophthalmology*, *Ophthalmic Year Book and Literature*. The editorial staff consists of Edward Jackson, editor, Clarence Loeb, associate editor, A. Alt, M. Uribe-Troncoso, M. Wiener, C. A. Wood, and H. V. Würdemann. There are many American and foreign collaborators. The journal will appear monthly, published by the Ophthalmic Publishing Co., 7 W. Madison Street, Chicago, Ill. The annual subscription price is $10.

The number of nearly 100 pages is wide in its scope, and comprises nine original and well-illustrated papers, several translations from foreign journals, short abstracts, society proceedings from Philadelphia, Colorado, and Chicago, editorials, book notices, news items, index of ophthalmic literature, and digest of the literature. The last part, the digest of the literature, is paged separately and ultimately will form, when bound together, the *Ophthalmic Year Book*. The size of the page in the new journal, the quality of the paper, and the type are all excellent; the lines are perhaps too close for comfortable reading.

The ARCHIVES welcomes the new journal with every good wish for its success.

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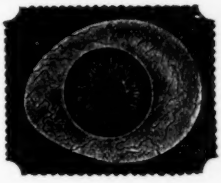

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